UNITED STATES DEPARTMENT OF AGRICULTURE
MISCELLANEOUS PUBLICATION No. 440

Washington, D. C. September 1941

MARKET DISEASES OF FRUITS AND VEGETABLES

ASPARAGUS, ONIONS, BEANS, PEAS, CARROTS, CELERY, AND RELATED VEGETABLES

BY

GLEN B. RAMSEY
Senior Pathologist

and

JAMES S. WIANT
Associate Pathologist
Division of Fruit and Vegetable Crops and Diseases
Bureau of Plant Industry

For sale by the Superintendent of Documents, Washington, D. C. - - - - Price 35 cents

UNITED STATES DEPARTMENT OF AGRICULTURE

MISCELLANEOUS PUBLICATION No. 440

Washington, D. C. September 1941

MARKET DISEASES OF FRUITS AND VEGETABLES: ASPARAGUS, ONIONS, BEANS, PEAS, CARROTS, CELERY, AND RELATED VEGETABLES[1]

By GLEN B. RAMSEY, *senior pathologist*, and JAMES S. WIANT, *associate pathologist, Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry*

CONTENTS

[1] This publication is the seventh in a series designed to aid in the recognition and identification of pathological conditions of economic importance affecting fruits and vegetables in the channels of marketing, to facilitate the market inspection of these food products, and to prevent losses from such conditions. It represents an extended revision and elaboration, with the addition of colored illustrations, of a preliminary publication entitled "Handbook of Diseases of Vegetables Occurring Under Market, Storage, and Transit Conditions," prepared by George K. K. Link and Max W. Gardner, and published by the Department of Agriculture in 1919 especially for the use of the food-products inspectors of the Bureau of Agricultural Economics and never distributed to the public. The material is organized on the basis of the botanical families to which the plants belong, but no botanical system is followed in arranging these families. Practical considerations make it desirable to issue the material in separate sections arranged somewhat in the order of the economic importance of the crops. The Host Index of the Fungi of North America, by A. B. Seymour, 1929, is used in the main as a guide to the nomenclature of causal fungi and the names of authorities therefor. The original colorings for plates 1, *A, B, C;* 3, *D;* 7, *C, D, E, G;* 10, *C, D, E;* and 12, *B,* are by Glen B. Ramsey. The photograph for plate 5, *F,* was furnished by J. C. Walker; 7, *E,* by Anna E. Jenkins; 8, *A, B,* by L. L. Harter; and 11, *E,* by W. C. Snyder.

303920°—41——1

For convenience in discussing the diseases common to a related group of plants, the botanical family is made the basis of the separate sections of this bulletin. Within the family the crops are listed in alphabetical order. The diseases described are likewise arranged in alphabetical order under each crop.

LILY FAMILY

The lily family (Liliaceae) is probably best known for its ornamental plants, although there are several important vegetables that belong to this group. Listed in the order of their importance on the market these food crops are onion, asparagus, garlic, leek, shallot, Welsh onion, and chive.

Asparagus

The vegetable known as asparagus is the young unbranched shoot of the asparagus plant (*Asparagus officinalis* L.). When cut just as it emerges from the ground the shoot is chlorophyll-free. The blanched asparagus shoots are used by canneries. If cutting is delayed until the shoot has been exposed to light chlorophyll develops, and the resultant stimulated growth leads to the production of green tender shoots, which are preferred on the market.

Among the factors that determine the marketability of asparagus are firmness, tenderness, and freedom from decay. Tenderness is primarily a matter of rate of growth and of age. Slow-growing shoots and shoots developed late in the growing season have well-developed vascular tissues and consequently are tough and woody. Lack of firmness is due to excessive loss of water, which, although it may take place before cutting, generally occurs afterward.

Some field diseases of asparagus influence the production seriously but seldom directly affect the marketable shoots. The most serious one is rust (*Puccinia asparagi* DC.). It so weakens the plant that the crown often becomes susceptible to attacks by soil fungi, and only a few weak shoots are produced the next year. Cercospora leaf spot (*Cercospora* sp.) causes only minor damage.

The roots and shoots may be affected by bacterial soft rot, fusarium rot (wilt), gray mold rot, phytophthora rot, and watery soft rot. Bacterial soft rot and fusarium rot are the most common causes of loss of asparagus during transit and marketing.

Although several insect pests may damage asparagus plants in the field, the feeding marks and eggs of the asparagus beetle are about the only injuries noted on the market.

(See *7*, *8*, *24*, *25*, *31*, *32*, *35*, *43*.)[2]

BACTERIAL SOFT ROT

(*Erwinia carotovora* (Jones) Holland)

Bacterial soft rot is the only decay of asparagus that has an odor. Like that of many other vegetables affected with bacterial soft rot, the odor is barely noticeable in the early stages of decay, but in the advanced stages it becomes very disagreeable.

Bacterial soft rot is most frequently found affecting the tips and cut ends of the shoots, but it may develop at any point where breaks in the epidermis occur (pl. 1, *B*). The bacteria responsible for this decay undoubtedly are more or less common in the soil and in water. They become destructive when high temperature and breaks in the protective cover of the tissues render conditions favorable for their attack.

The development of bacterial soft rot in asparagus during transit may be controlled by discarding all badly bruised and crushed shoots and by immediately precooling the marketable stock to about 40° F., so that the transit refrigeration provided will be able to maintain low temperatures throughout the trip.

For a detailed discussion see Celery, Bacterial Soft Rot, p. 46.

BEETLE INJURY

The asparagus beetle (*Crioceris asparagi* (L.)) is a very important cause of injury to asparagus on the market. The insect overwinters in plant rubbish in the adult or beetle stage and emerges early in the season to feed upon the young shoots and deposit eggs upon them. The small black eggs are deposited in great numbers upon the young shoots and are very hard to remove. The presence of these eggs upon the shoots greatly reduces the market value of asparagus. The feeding marks of the beetle may also blemish the shoots and at times open the way for the entrance of decay-producing bacteria and fungi.

FUSARIUM ROT (WILT)

(*Fusarium* spp.)

Species of *Fusarium* often invade the tips and other parts of tender asparagus shoots, causing considerable loss in shipments to distant markets. These fungi first appear as a white fluffy mold, which later may become slightly pink (pl. 1, *C*). The affected tissues are watersoaked at first, later changing to yellow and brown. Side lesions sometimes extend 2 or 3 inches lengthwise of the shoots. Affected tips may become completely covered with mold and the tissues converted into a soft watery mass. No odor accompanies this decay unless bacteria also are present.

[2] Italic numbers in parentheses refer to Literature Cited, p. 60.

The causal organisms live in the soil and are present on the asparagus shoots when they are cut and packed for shipment. At least two species of *Fusarium* have been isolated from decaying shoots and from wilted, stunted, and yellowish plants in the field. Whether these or other species cause the moldy tips and decay during transit is not yet known.

Test shipments under different methods of precooling and refrigeration and laboratory temperature studies have shown that the decay-producing organisms are held in check satisfactorily at a temperature of 40° F. Affected stock develops decay rapidly, however, soon after it becomes warm.

(See *11.*)

GRAY MOLD ROT

(*Botrytis* spp.)

Although the organisms that cause gray mold rot are ever present in most vegetable-growing regions, they seldom cause serious injury to asparagus. Under conditions favorable to them, however, they have been known to cause a tip wilt and decay in the field and during transit. The water-soaked lesions usually show a small amount of white surface mold in the early stages; later, the grayish-brown mycelium and granular spore masses serve to identify the disease.

For a more complete discussion of gray mold rot, see Carrot (p. 41) and Peas (p. 34).

PHYTOPHTHORA ROT

(*Phytophthora* spp.)

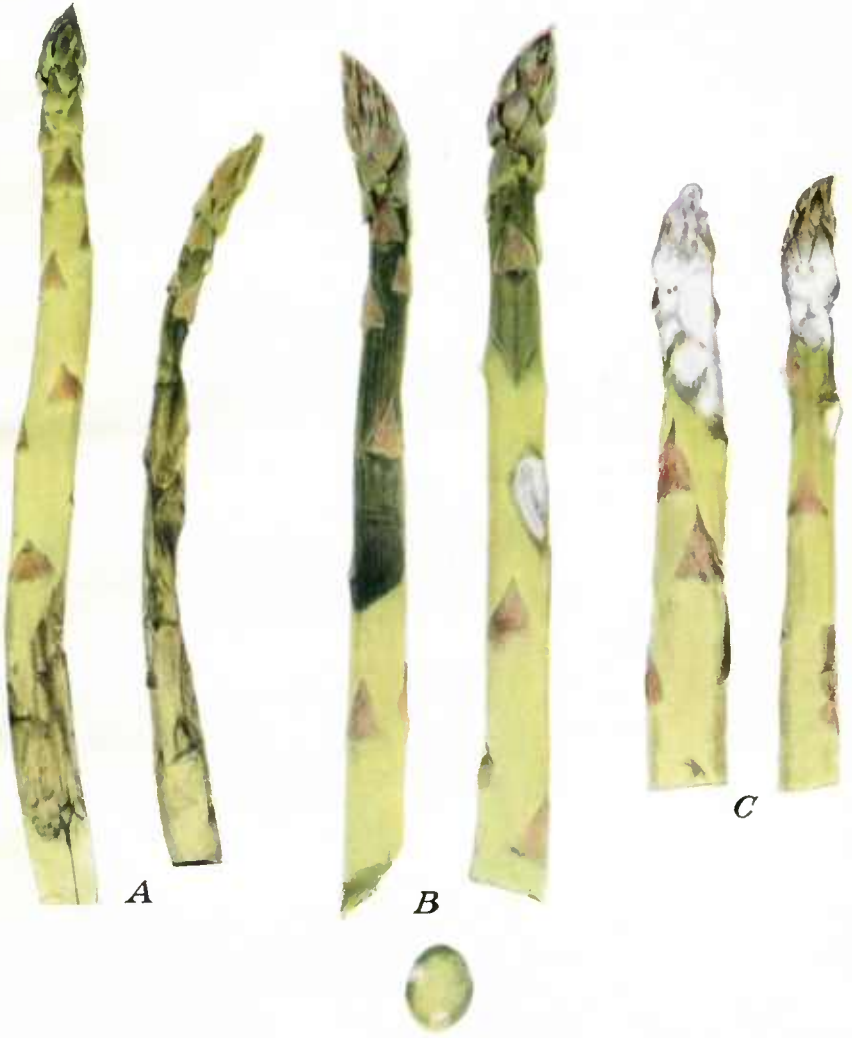
A serious decay induced by a species of *Phytophthora* occurred in shipments of California asparagus during the seasons of 1938 and 1940.[3] This disease had not previously been found affecting asparagus on the market.

The decay is characterized by large semiwatery, light-brown lesions generally occurring on the side of the shoots about 1 or 2 inches above the base (pl. 1, *A*). At times the decay completely encircles the shoot, but most often one side only is affected and the broken-down tissues collapse, causing an infolding and shriveling that result in the production of a flattened or creased side to the shoot. Under moist conditions a grayish-white, wet, appressed mold is evident on the surface of the larger lesions, but under dry conditions there may be no surface mold apparent. The decaying shoots have no odor unless bacteria are also present.

As this soil fungus usually causes no serious damage except under excessively wet conditions, it is not probable that the disease will commonly occur on asparagus. The California asparagus fields were flooded in the springs of 1938 and 1940 when this disease caused so much decay in the field and during transit and marketing.

(See *5.*)

[3] WIANT, JAMES S., and BRATLEY, C. O. DISEASES OF FRUITS AND VEGETABLES ON THE NEW YORK MARKET DURING JANUARY, FEBRUARY, MARCH, AND APRIL 1938. U. S. Bur. Plant Indus., Plant Dis. Rptr. 22: 190–193. 1938. [Mimeographed.]



A, Asparagus phytophthora rot. *B*, Asparagus bacterial soft rot. *C*, Asparagus fusarium rot.

WATERY SOFT ROT

(*Sclerotinia sclerotiorum* (Lib.) DBy.)

Watery soft rot of asparagus in early stages is similar in appearance to fusarium rot but is of less common occurrence. The later stages can usually be distinguished from fusarium rot by the more luxuriant white cottony mold, the presence of sclerotia, and the fact that the mold never turns pink.

Affected tissues are watery, odorless, and characterized by the presence of the cottony, white growth of the mold. The decay causes losses both in the field and during the marketing process. Contaminated soil and water, or infected shoots packed unknowingly, are the source of the decay that develops during transit. Neither the fungus nor the lesions need be visible on the product when it is packed in order for decay to develop during transit.

For a detailed discussion of the cause and relation of environmental factors and control, see Celery, Watery Soft Rot, page 56. (See 204.)

GARLIC AND ONIONS

Garlic (*Allium sativum* L.), onion (*A. cepa* L.), and their close relatives, leek (*A. porrum* L.), chive (*A. schoenoprasum* L.), shallot (*A. ascalonicum* L.), and the Welsh onion(*A. fistulosum* L.), all belong to the lily family. They are used as food, both fresh and cooked, and for flavoring and seasoning.

The plants consist of long, slender, erect leaves that arise from an extremely short stem or scale plate, from the lower side of which originate numerous fibrous roots. Toward the close of the growing period, the terminal bud develops into a long, slender, hollow flower stalk, on which either flowers and seed or "top onions," or bulblets, are produced. In the onion, leek, shallot, and garlic the leaves or scales have a tendency to be fleshy near the scale plate, thus giving rise to a bulb. In the onion, the outer dry papery scales of the bulb portion may be white, red, yellow, or brown, depending upon the variety and these afford the bulb protection from drying out and from attack by parasites. The tops of the fleshy scales are green. When onions are grown for table use as green onions, they are pulled before the bulb develops. In certain species of *Allium* there is a decided tendency toward proliferation at the base so that a cluster of small bulbs results, as in shallots, garlic, or the multiplier onion. Onion sets are produced by arresting the growth of the young plant or from the bulblets of "top onions" or of "multipliers."

Firmness of the bulb is an important factor in the marketability of onions and the bulbous members of the onion group. Lack of firmness generally indicates incomplete development. This may result from unfavorable soil conditions, especially lack of water, from root diseases, or from premature death of the tops. The last may result from unfavorable climatic factors, insect and fungus attack, or premature topping. Immature bulbs tend to dry out and shrivel abnormally after being harvested.

The onion and its relatives are subject to a considerable number of diseases. Some of these occur on the plant in the field, affecting the

yield or the quality, or both; others are important primarily in storage and transit; and some are factors both in the field and on the market.

Among the serious field diseases are downy mildew (*Peronospora destructor* (Berk.) Caspary), fusarium bulb rot, pink root, purple blotch, smut, and yellow dwarf (virus). Less important are root knot (*Heterodera marioni* (Cornu) Goodey), rust (*Puccinia asparagi* DC.), and white rot.

Several serious diseases belong in the group that are primarily important from the marketing point of view. These are bacterial soft rot, black mold rot, blue mold rot, gray mold rot, and smudge.

Greening (sunburn) and sunscald occur in the field, sometimes causing serious damage at harvesting time and later during the marketing process.

Chemical injuries (ammonia injury and "alkali spots") are primarily transit and storage troubles.

(See *6*, *10*, *16*, *17*, *22*, *23*, *27*, *50*, *52*, *57*.)

Garlic (*Allium sativum* L.) is subject to many of the diseases that affect the onion. Aspergillus rot (p. 7), fusarium rot (p. 11 and pl. 2, *C*), gray mold rot (p. 12), sclerotium rot (see sclerotium rot of beans, p. 30) and white rot (p. 17) are among the most important diseases of the bulbs. However, the diseases most often encountered on the market are blue mold rot and waxy break-down.

BLUE MOLD ROT OF GARLIC

(*Penicillium* spp.)

Blue mold is not often of serious consequence on vegetable crops, but occasionally it causes appreciable decay of garlic and onion bulbs. Stock that shows excessive mechanical injury, freezing, or sunscald is especially subject to blue mold rot during storage or transit if there is insufficient ventilation.

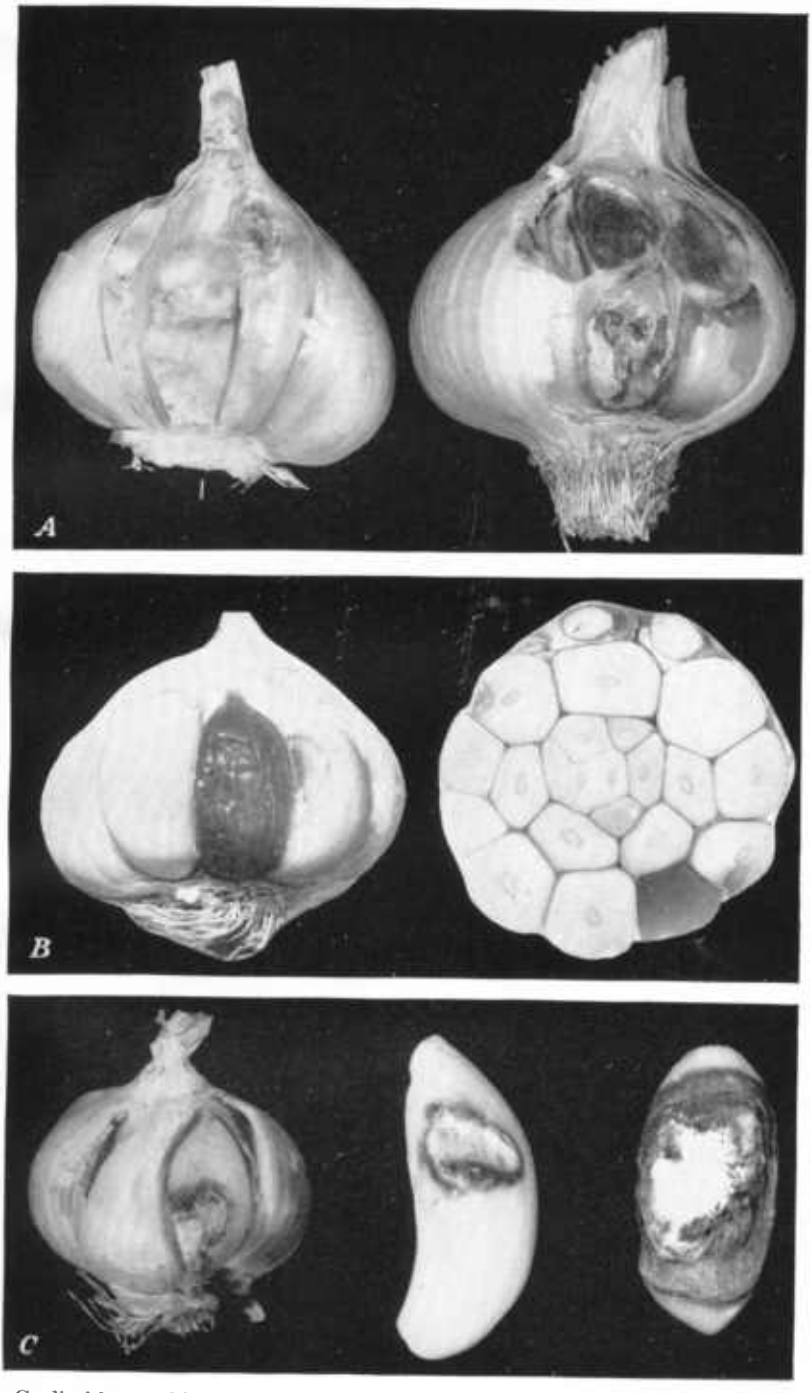
The early symptoms of blue mold rot are light-yellowish lesions in the fleshy scales of the bulbs. As the decay progresses a fine white surface mold becomes visible (pl. 2, *A*), but this soon changes to blue or bluish green as spores are produced in great abundance on the tips of the fungus filaments. The appearance of this characteristic blue mold serves to identify readily the disease.

Garlic bulbs affected with blue mold rot may show little or no evidence of the disease externally. However, affected bulbs will be light in weight, and pressing on individual cloves will reveal a soft, spongy or powdery-dry condition, depending upon the extent of the decay. In advanced stages of decay the cloves are completely broken down into a dry greenish-tan or gray powdery mass.

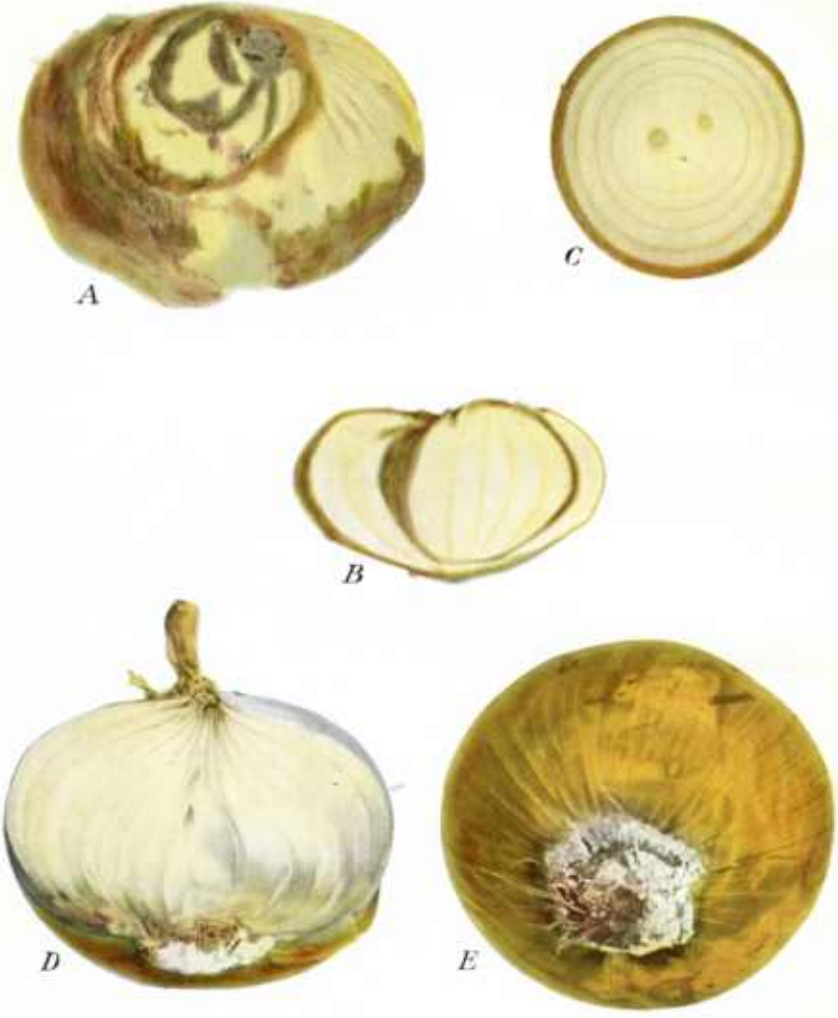
No data are available regarding the rate of development of blue mold rot in garlic during the curing process or while in storage.

WAXY BREAK-DOWN OF GARLIC

A yellow waxy break-down of the outside cloves of garlic bulbs is one of the serious troubles that affects the marketability of this crop. Little is known as to the cause of this break-down except that no organism seems to be associated with it as a causal agent.



A, Garlic blue mold rot. *B*, Garlic waxy break-down. *C*, Garlic fusarium rot.



A, *B*, Onion bacterial soft rot. *C*, Onion freezing injury. *D*, *E*, Onion fusarium bulb rot.

Whether it is due to high temperature, sunscald, or some physiological disturbance is not known.

The early symptoms of waxy break-down are small, slightly sunken, light-yellow areas in the flesh of the clove. As the break-down progresses the clove shows a deep-yellow or amber color throughout (pl. 2, *B*). The clove is then somewhat translucent and sticky or waxy to the touch but not soft, as in the case of tissues broken down by parasitic organisms. The outer dry protective scales are not affected. No indications of break-down may show externally until the advanced stage is reached, when the shrinking of the clove and the amber-brown color may become noticeable through the white papery outer scales.

ASPERGILLUS BULB ROT OF ONIONS

(*Aspergillus alliaceus* Thom and Church)

Although aspergillus bulb rot has not yet been found in the United States, the causal fungus has been intercepted at ports of entry in garlic from Italy. As inoculation tests have proved that this fungus is pathogenic to American onions and garlic, it may become of economic importance in this country.

On inoculation of garlic and mature onions the first sign of disease is a slight swelling and a darkening of the tissue. This affected area becomes water-soaked and soft, and a sharp line of demarcation occurs between the diseased and the healthy tissue. In advanced stages the tissue shrinks and becomes brownish and a white mat of mycelium forms between the bulb scales. Eventually, small white sclerotia form, which later become dark brown at maturity. The spore clusters appear yellowish brown and are produced in great abundance on the surface of the decaying tissue if the humidity is high.

Infected bulbs are completely decayed within 21 days when held at 96 8° F. Practically no decay develops in inoculated bulbs kept for 3 months at 60.8°. The results of attempted inoculations of growing plants indicate that the fungus is not able to attack them and that it may become important only as a decay-producing organism of the matured bulbs.

No control measures have been developed.

(See *54*.)

BACTERIAL SOFT ROT OF ONIONS

(*Erwinia carotovora* (Jones) Holland)

Bacterial soft rot occurs in all onion-growing districts in the United States but is most common in onions harvested during warm, rainy seasons or in those that develop sunscald during the curing process. It is often serious in imported Spanish onions, especially after the sprouting tops are cut off.

This decay occurs in the field, where it causes some losses, and in transit and storage, where the losses frequently are very heavy. The affected tissues are glassy or water-soaked at first, later becoming soft and mushy (pl. 3, *A*). The rot usually is accompanied by a very repulsive odor. The bacteria that cause it apparently cannot penetrate the dry unbroken skin of the onion. In the field they generally

enter through the neck. Entering this way either in the field or after topping, the decay progresses downward from the neck and often is confined to one or two scales in the interior or to the central scales (pl. 3, *B*). When this occurs the only external sign of the disease is the loss of firmness, which can be detected by pressing the bulb. This often forces a mushy, foul-smelling mass out at the neck. Any injury on the onion, such as sunscald, freezing, and wounds due to careless handling, may open the way for this rot.

See Celery, Bacterial Soft Rot, page 46.

(See *34*.)

BLACK MOLD ROT OF ONIONS

(*Aspergillus niger* Van Tiegh)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The black mold rot occurs on all varieties of the onion. Although it attacks bulbs in the field it does not affect the yield. If rainy weather prevails when the onions are pulled, the disease may seriously blemish the stock, even though only the outer dry and dead scales are involved. Under moist conditions in transit or storage the fungus grows actively and rotting results; consequently the disease is of considerable importance from the market point of view (pl. 4, *C*).

Black mold rot may be a serious factor as a blemish or as a decay of onions from any section of the country. However, it seems to be a market factor more frequently in Texas and California stock than in onions from other sections.

The chief sign of this disease is the presence of black, powdery spore masses of the fungus on the outside scale or between that and the next inner one. When these masses occur between the scales they tend to follow the veins (pl. 4, *A*, *B*). Sometimes there are no apparent lesions and the fungus itself constitutes the only blemish. At times the tissues show symptoms, such as sunken discolored areas, which underlie the powdery fungus masses. Under dry conditions the affected tissues are dry and papery and sometimes highly colored; under moist conditions they are semiwatery.

CAUSAL FACTORS

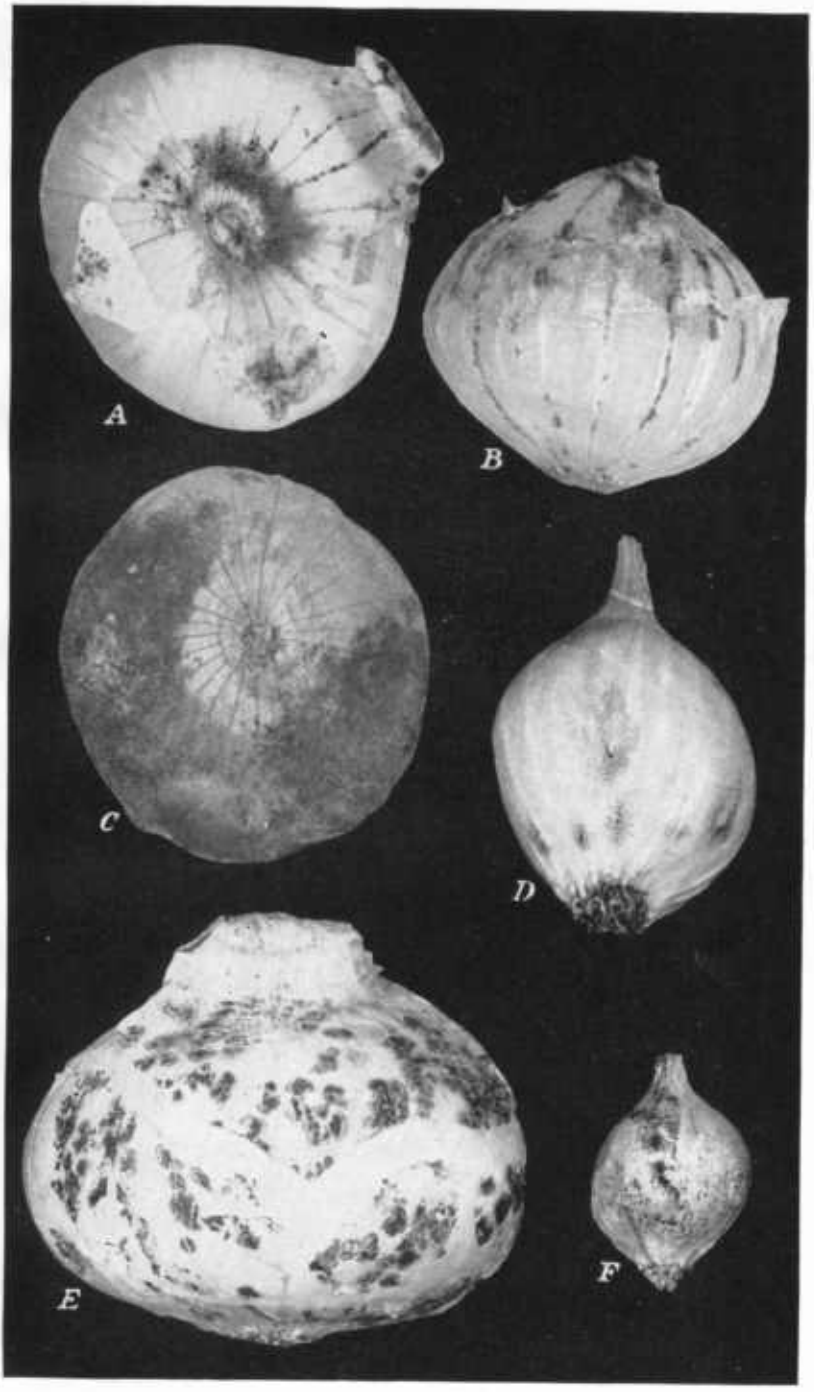
The pathogen, *Aspergillus niger*, is one of the soil fungi that live on dead or dying plant material. From the soil it spreads to the growing onion, attacking the outer scales and neck tissue. In transit and storage it may spread from onion to onion by contact through bruises and wounds, or by dissemination of the spores by air currents and by mechanical means.

Observations in the field, in storage, and in transit indicate that moisture and high temperature favor the growth and spread of the fungus. It grows slowly at 55° F. and makes most rapid development at about 95°.

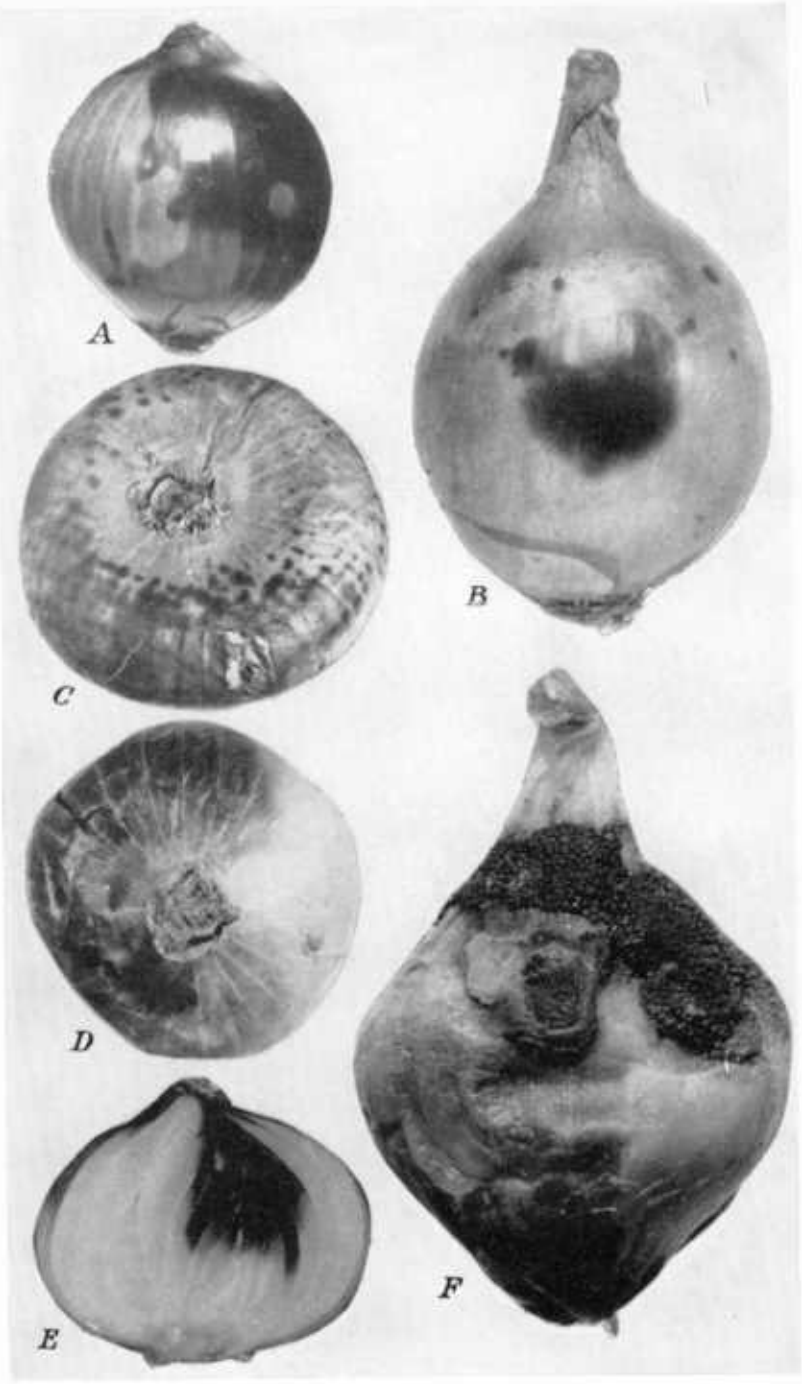
CONTROL MEASURES

Protection from moisture in the field during and after pulling and during transit and storage is advisable. The disease develops and spreads less rapidly in cold storage than in common storage.

(See *20*, *40*, *50*.)



A, *B*, *C*, Onion black mold rot. *D*, *E*, *F*, Onion smudge.



A, Onion ammonia injury. *B*, *C*, Onion alkali spot and bag print. *D*, *E*, Onion purple blotch. *F*, Onion white rot.

BREAK-DOWN OF ONIONS

This trouble is encountered in onions from various regions. It is often so important in storage stock that many refer to the injury as "storage break-down," but investigations have shown that it also occurs in onions taken directly from the field.

This break-down is characterized by the grayish water-soaked appearance of the outer fleshy scales of the bulbs, and is similar to some cases of freezing injury. In fact the symptoms are so alike that an accurate diagnosis of these two troubles is often not possible until a complete history of the stock is known. However, in physiological break-down the outer two or three fleshy scales are usually the only ones affected; the water-soaked tissues may not extend completely around the bulb; and they do not contain the opaque areas often found in frozen onions.

The cause of this type of break-down is not known. Apparently certain growing conditions favor the development of this injury in the field and later in storage. Some experiments have indicated that break-down is more likely to occur in storage onions at high humidities and temperatures of 40° to 50° F. than at lower humidities and temperatures near 32°. A relative humidity of about 64 percent is probably best for most onions, because it prevents root growth that is likely to occur at higher humidities.

(See *57*, *58*.)

CHEMICAL INJURY OF ONIONS

One of the most common types of discoloration of onions caused by chemicals is illustrated by the "scorched spot," "alkali spot," and "bag print" blemishes sometimes found on colored onions that have been in contact with moist bags or in damp storage for some time. Small oval spots varying in size from ½ to 1 inch in diameter are produced on the sides of the onions that come in contact with the fabric (pl. 5, *B*). Sometimes the spots are uniformly dark-brown or black, as if the onions had been scorched, and sometimes the mesh of the bag is distinctly printed in dark, brownish-black on the onion scales (pl. 5, *C*). This discoloration is caused by a chemical in the fabric of the bag that produces an alkaline reaction with the color pigments of those scales in contact with the fabric. If the bag is moist, the discolored areas are uniformly brown or black; if it is comparatively dry the diffusion of the chemical is slight, and the color change takes place so slowly that the imprints of the individual meshes of the fabric become prominent. With a discoloration of this type, however, no injury to the underlying fleshy scales that would impair the eating qualities of the onion has ever been observed.

Not all onion bags give this chemical reaction; consequently such blemishes are likely to be more or less sporadic and generally are not serious.

A more uniform and severe type of discoloration caused by a chemical is occasionally found in onions held in cold storage (pl. 5, *A*). This injury is caused by the accidental exposure of the stock to ammonia fumes from the storage plant. The fumes cause an alkaline reaction with the color pigments in the outer scales of the onions and often produce such pronounced discoloration that the marketability of the stock is greatly reduced. As a result of

exposure to ammonia fumes yellow onions show brown blotches or a uniform brown color over all the exposed surfaces; red onions change to a deep greenish-black or metallic-black color, and white onions are discolored greenish yellow. If onions are exposed to strong fumes for several hours the fleshy, edible portion of the bulbs becomes watery, yellowish green, and worthless.

Experiments have shown that ammonia injury will take place at a temperature as low as 31.5° F., and that the rate and severity of discoloration are approximately the same as at higher temperatures. The color changes have been found to take place more rapidly in a humid atmosphere or when the onions are slightly moist. Less than 1 percent of ammonia in the air will cause marked discoloration if the onions are exposed for 24 hours or longer. With stronger concentrations the color changes are noticeable almost immediately and large brownish-black blotches are produced within a few minutes.

A similar type of discoloration in colored onions has been observed in stock that had been covered with manure for protection against freezing. The ammonia fumes from the manure cause almost a complete blackening of the outer dry colored scales but generally do not discolor the fleshy scales.

(See *27*, *28*, *36*.)

FREEZING INJURY OF ONIONS

The average freezing point of onion bulbs is about 30° F. Although some onions may freeze at a fraction of a degree higher or lower, this temperature may be taken as the danger point. Under some conditions onions standing on track or in storage may be undercooled to a temperature of 25° or lower for a short time without becoming frozen or otherwise injured. A slight jar will cause undercooled products to freeze immediately; consequently undercooling rarely occurs during transit.

There is considerable variation between individual onions in their reaction to low temperature. Some bulbs in a lot may freeze quickly when they reach a temperature of 30° F. and show severe injury when they are thawed out. Others may not freeze or if they are frozen they may thaw out without injury.

Onions affected with freezing injury show water-soaked, grayish-yellow colored fleshy scales when cut (pl. 3, *C*). In slight freezing the outer fleshy scales alone are affected, but when the bulbs are exposed to low temperatures for a prolonged period the inner scales may also become water-soaked and discolored. Usually the entire scale is injured all the way around the bulb, but the neighboring scales inside and outside may or may not show injury. Irregularly shaped opaque areas occur in many of the watery transparent scales.

Garlic bulbs do not freeze until a temperature of 25.4° F. is reached. The frozen tissues are discolored and water-soaked in the same way as onions.

In cases of slight freezing injury of onions and garlic there may be little loss if the bulbs are spread out so that the injured scales can dry thoroughly. Experimental evidence indicates that frozen onions will show less injury and remain in a better general condition if thawed out at 40° F. rather than at a higher temperature.

(See *19*, *57*, *58*.)

FUSARIUM BULB ROT OF ONIONS

(*Fusarium oxysporum* Schl. f. 7 Wr., *F. vasinfectum* Atk. var. *zonatum* (Sherb.) f. 1 and f. 2 (Lk. et Bail.) Wr.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Fusarium bulb rot of onion is of great importance from both the production and the marketing point of view. The rot causes reduced yields in the field, and in transit and storage it is responsible for heavy losses. It has been reported from practically all onion-growing sections of the United States and is especially severe in the warmer sections. On the market it has been found serious in shipments from Ohio, Illinois, Wisconsin, Colorado, California, and Washington.

The first symptoms of bulb rot visible in the field are the yellowing and rapid dying of the leaves from the tips down, at about the time of maturity. The advancing edge of the lesion may be somewhat yellow, but the dead leaves have a silvery-gray appearance. However, these symptoms do not invariably indicate bulb rot; in fact, the rot cannot be identified positively until the onion is pulled. Affected onions pull easily because many of the roots are rotted off. The base of the onion may be covered with a dense growth of white to pinkish mold, and in severe cases the bulb may show a semiwatery, a mealy, or a dry decay which progresses along the scales from the base upward (pl. 3, *D*, *E*). Whether the rot is dry or watery usually is determined by the species of *Fusarium* causing the rot, and also by moisture and temperature conditions. High moisture and high temperatures tend to produce a dry rot. Storage onions in advanced stages of decay may be converted into shriveled mummies. The diseased bulbs sprout much more freely than healthy ones.

CAUSAL FACTORS

Although various species of *Fusarium* alone or in association with bacteria are able to induce bulb rot, detailed study indicates that *Fusarium oxysporum* f. 7 and *F. vasinfectum* var. *zonatum* f. 1 and f. 2 are the most important. These fungi live in the soil and from this they attack the growing bulb. They also overwinter in infected bulbs and probably can live over in debris and containers in storage houses. Continuous cropping with onions increases the amount of inoculum in the soil.

Field observations and experimental work with these species indicate that high temperatures favor the growth of the pathogens, and the inception, spread, and development of the disease. In the field these organisms cause rot at temperatures between 59° and 89° F., developing most rapidly between 82° and 89°. In storage there is little decay at temperatures between 46° and 59°, but there is a pronounced tendency toward premature sprouting in affected bulbs at these temperatures. At 68° the rot progresses rapidly but the tissues remain watery for a long time; at 86° the tissues decay and dry out rapidly. The rot develops and spreads in transit and storage. Once it has become established, there is a possibility of its spreading through the whole lot, provided temperature and moisture conditions are favorable. Wounds and insect injuries favor the development of rot because they make entrance easier for the organisms.

CONTROL MEASURES

It is not advisable to plant the same field to onions year after year. Crop rotation is especially beneficial in regions where bulb rot is likely to become severe. Affected stock should not be put in storage or shipped long distances but should be put into immediate consumption. If necessary to store diseased stock the development of decay will be retarded by temperatures below about 45° F.

(See *13*, *14*, *17*, *55*, *56*.)

GRAY MOLD ROT (NECK ROT) OF ONIONS

(*Botrytis allii* Munn., *B. byssoidea* Walker, and *B. squamosa* Walker)

OCCURRENCE, SYMPTOMS, AND EFFECTS

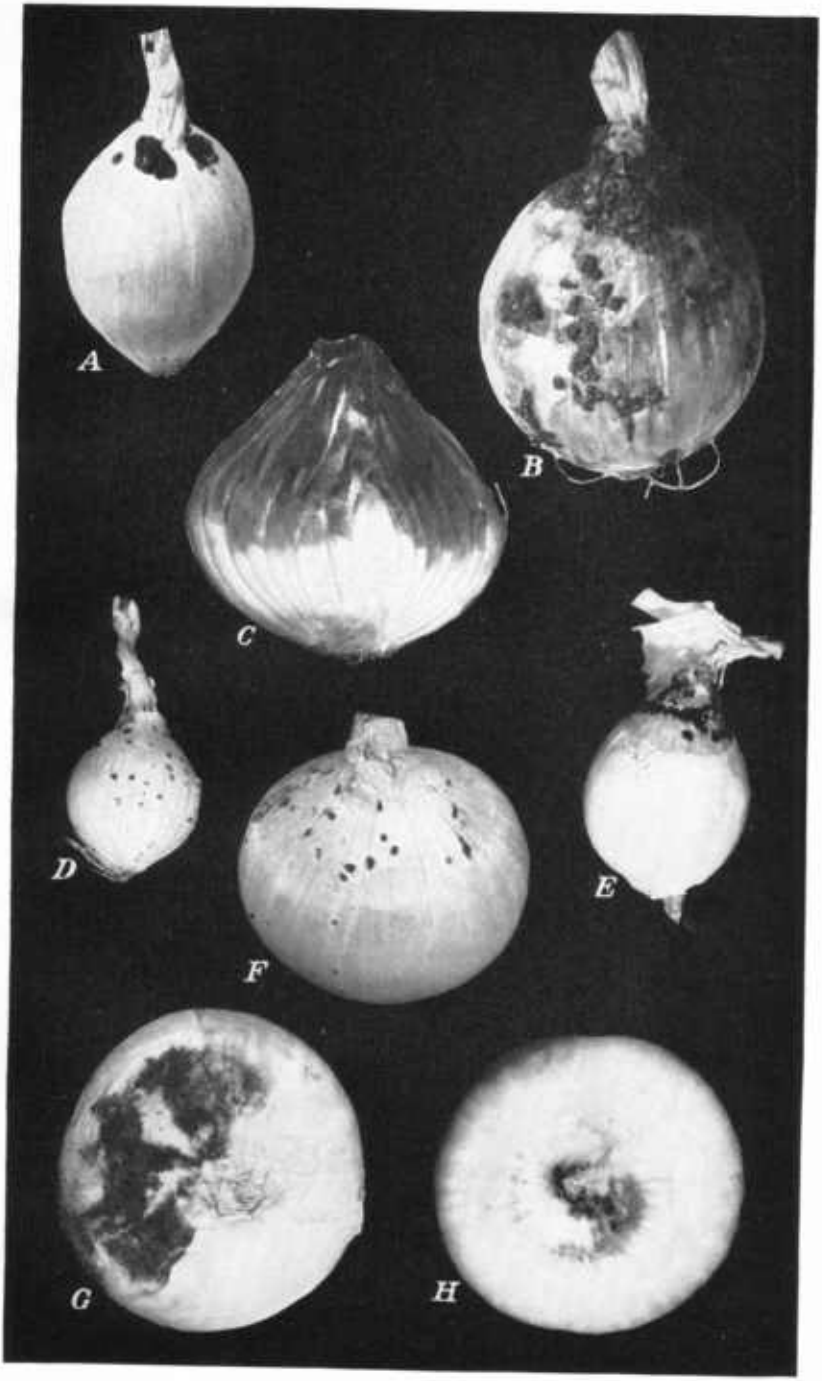
Although gray mold rot causes practically no damage to the growing crop, it is one of the most serious troubles affecting onions following harvesting and during transit and storage. It occurs to some extent in all onion-growing regions and frequently is the limiting factor in determining the marketability of a crop. At times 50 percent or more of a crop is lost through the development of neck rot during storage. Apparently all varieties of onion, shallot, and garlic are susceptible to this disease, but the white ones are the most seriously affected.

Infection usually takes place through the neck tissues of the bulbs at harvesttime. This characteristic mode of infection has led to the use of the term "neck rot" for describing this disease, although frequently infections take place through wounds made in the base or sides of the onions.

The first indication of the disease is a softening and water soaking of affected scales. Soon this tissue becomes grayish and finally grayish brown as the mycelium of the fungus develops within (pl. 6, *B*, *C*, *E*, *H*). Later, a dense layer of white to grayish mold develops over the surface of the affected region. On the mold growth in the older lesions on the outside scales and between the scales at the neck, fine grayish-brown, powdery masses of spores are borne (pl. 6, *G*, *H*). This development of gray mold and grayish-brown spores has given rise to the term "gray mold rot" as a designation for this decay, particularly when it occurs at the base or sides of the bulbs and not at the neck. The decay develops laterally from the point of infection, as well as down through the fleshy scales. The line of demarcation between the diseased and healthy tissue is quite definite, although the advancing edge of the lesion usually shows a slight water soaking of the fleshy tissues. No odor accompanies this decay. In advanced stages of decay the affected tissues become sunken and fairly firm. In stored onions that have developed advanced decay at the neck, there may be found firm black bodies (sclerotia) 1/32 to 1/4 inch in diameter on the decaying tissues (pl. 6, *A*, *B*).

CAUSAL FACTORS

Three closely related species of *Botrytis* cause types of gray mold rot or "neck rot" that are somewhat similar. The rot caused by *B. allii* is probably the most common form (pl. 6, *G*, *H*). It is charac-



Onion gray mold rot: *A*, *B*, *C*, *Botrytis byssoidea* type. *D*, *E*, *F*, *Botrytis squamosa* type. *G*, *H*, *Botrytis allii* type.

terized by its compact gray-brown mycelium and abundant development of spores, and often by the formation of crustlike sclerotia about the necks of storage onions. The decay caused by *B. byssoidea* (pl. 6, *A*, *B*, *C*) cannot easily be distinguished from the first-mentioned form. It differs chiefly in the production of a greater abundance of light-gray fluffy mycelium. The rot caused by *B. squamosa* has been found on the market only on white onions. The mycelium and spores are inconspicuous, and the first evidence of the disease is often the appearance of very thin small black sclerotia adhering to the dry outer scales at the neck of the onion (pl. 6, *D*, *E*, *F*). The decay is much slower in development than that induced by the other two organisms, and when advanced stages are reached the affected tissues are darker brown than in either of the other types of gray mold rot.

Each of the neck rot organisms is able to grow throughout a wide range of temperature. Below 37° and above 91° F. their growth is very slow. At 68° to 77° most rapid growth is made in pure culture, but inoculation experiments have shown that temperatures between 59° and 68° favor the development of decay. High humidity favors infection and the spread of decay from one bulb to another by contact in storage. Cool, wet or humid weather favors the development and spread of the fungus in the field, particularly just previous to and during harvesting time. Hot, dry weather not only checks the growth of these fungi but it also causes the onions to mature more rapidly, so that the necks cure quickly and become dry before infections take place.

These fungi live from season to season as sclerotia in the soil and as sclerotia and mycelium on infected bulbs. They also live on dead onion leaves and other plant debris during the growing season. The bulbs become infected by wind-borne spores at maturity and during the harvesting and curing processes.

CONTROL MEASURES

If the weather permits a satisfactory maturing of the crop and prompt thorough curing at harvesting time, the neck rots caused by *Botrytis* species are easily controlled. When the onion necks are moist and curing is slow and unsatisfactory, the application of warm air by artificial means has been found advantageous in rapidly drying the neck tissues.

Because colored varieties are less susceptible to neck rot than white ones, a change in varieties may be advisable in some localities.

Onions should be kept in a cool, dry storage. Even cold-storage temperatures of 32° F. will not completely check the development of gray mold rots, if the bulbs are already infected when placed in storage.

(See *18*, *21*, *41*, *48*, *49*, *50*.)

GREENING (SUNBURN) OF ONIONS

One type of injury caused by sunlight is known as greening or sunburn. It is caused by exposure of the bulb to moderate light during the growing period or after harvesting. The sunlight causes a development of green coloring matter in the exposed tissues but

does not cause death or softening of the affected area. Green bulb tissues are bitter and unpalatable, but as there is little danger of decay following, the marketability of onions so affected is not greatly influenced unless the lot as a whole shows this blemish quite prominently.

PINK ROOT OF ONIONS

(*Phoma terrestris* Hansen)

Pink root has been found affecting onions, shallots, garlic, leeks, and chives. The disease is common to many onion-growing sections, but the greatest damage occurs in the Texas, Louisiana, Iowa, and California crops. Onions and other plants belonging to this group are subject to attacks by the causal organism in all stages of development from the seedling stage to maturity. Although primarily a field trouble, this disease is also of market importance because it causes a reduction in the size of the bulbs and the lesions produced open the way for *Fusarium* species that often cause serious decay of the bulbs in the field and during the marketing process. (See Fusarium Bulb Rot, p. 11.)

Plants affected by pink root usually show symptoms characteristic of starvation or drought injury. The early symptoms noted on the roots are a collapsing of the tissues, shriveling, and discoloration varying from pink to dark-red and purplish hues. Many plants affected with pink root show no symptoms other than the pink discoloration of the roots. Such plants have developed new roots fast enough to permit continued growth and bulb formation. No decay of the bulb occurs when only the pink root fungus is present.

The causal organism persists in the soil from season to season. The minimum temperature for its development is 55° F.; the optimum is about 77° to 80°; and the maximum is above 90°.

No satisfactory control measures have been developed for pink root. In tests for resistance to the disease it has been found that most varieties of the common onion, garlic, and shallot are extremely susceptible. Chives, leeks, and Welsh onions (Nebuka type) are very resistant.

(See *13*, *14*, *15*, *26*, *30*, *37*.)

PURPLE BLOTCH OF ONIONS

(*Macrosporium porri* Ell.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Purple blotch disease has been reported from time to time on onions from various regions, but usually it is not of great economic importance except under very favorable weather conditions. Leaf mold (*Thyrospora parasitica* (Thüm.) Angell) and downy mildew (*Peronospora destructor* (Berk.) Caspary) of onions are so commonly associated with this disease that frequently the activities of the blotch organism are obscured. On the stems and leaves small grayish sunken lesions become visible within 4 days after inoculation; shortly thereafter small dark spots appear in the centers, later enlarging into purplish areas. Infection of the bulbs occurs at harvesting time, usually at the necks

of the topped bulbs or through wounds made elsewhere on the bulbs. These lesions are semiwatery at first, becoming papery as they dry. The color of the lesion is the most characteristic symptom. The fungus appears to secrete a pigment that diffuses through the tissues in advance of the hyphae. These tissues are yellow at first, later becoming red, then brown, and finally almost black as the dark-colored hyphae fill the older parts of the lesions (pl. 5, *D*, *E*). Sometimes only one or two outer scales are affected whereas at other times the entire bulb may be destroyed, especially in the case of winter bottom sets.

CAUSAL FACTORS

The causal organism develops most rapidly between 72° and 86° F. Very little growth takes place above 93° or below 50°. At temperatures of 70° to 72°, bulbs may be completely rotted in about 2 weeks.

CONTROL MEASURES

In regions where this disease is likely to become important, control may be effected by thorough and rapid curing and drying of the bulbs after harvest. Onions should be stored in a dry, well-ventilated house at temperatures between 33° and 36° F.

(See *3*, *9*, *38*, *44*.)

SMUDGE (ANTHRACNOSE) OF ONIONS

(*Colletotrichum circinans* (Berk.) Vogl.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Smudge attacks the onion, garlic, and shallot. Although it occurs in the field it does not reduce the yield. Its importance lies in the fact that it attacks onions at harvesttime and blemishes the scales, thus decreasing the quantity of marketable bulbs. The injuries caused in the field increase in transit and storage, with this addition, that under very moist conditions the fungus not only causes blemishes but also leads to destruction of the scales with consequent shrinkage. The disease also causes sprouting of sets in storage.

Smudge is characterized by black blotches or aggregations of minute black or dark-green dots on the outer scales (pl. 4, *D*, *E*, *F*). Each of these dots is a fruiting body (acervulus) in which are borne innumerable spores of the fungus. These dots often are in concentric rings, though they may be grouped in other ways. In severe cases the smudgy spots are so extensive that the side of the onion appears smoked. Usually the lesions are on the outer scales, but they may appear on the inner ones as well. On fleshy scales the fungus produces sunken, yellowish spots that enlarge slowly. The disease rarely occurs on yellow and red varieties and then only on the uncolored portions of the outer scales at the neck of the bulb. The disease occurs widely, but only white varieties are seriously affected.

CAUSAL FACTORS

The pathogen is the fungus *Colletotrichum circinans*. It occurs on onion scales in the soil or on bulbs and sets in storage, which carry

it into the field. The disease appears shortly before harvest. The spores, which are borne in abundance in the acervuli, are carried in drops of water to other scales where they germinate in a few hours, starting new lesions. If the weather is warm and moist, the fungus enters the onion scale and produces a new crop of spores in a few days. If favorable conditions continue, a relatively small amount of disease in the field before harvest will supply enough spores to blemish a crop severely during harvesting or while the crates are stacked in the field.

The fungus grows throughout a wide temperature range (36° to 89° F.) but develops best at about 78°. The spores germinate at temperatures ranging from 40° to 89°, the optimum lying between 68° and 78°. Infection may occur at 50°, but development is very slow below 68°. The disease develops and spreads most rapidly in the field when the soil temperature ranges between 68° and 86° and when there is an abundance of rainfall. Under favorable conditions the spores germinate and penetrate the scales in less than 24 hours.

CONTROL MEASURES

Except for planting colored varieties, which are highly resistant, no measures have been worked out thus far for control of the disease in the field. Control of the disease in the harvested crop depends upon protection of the onions from rain, rapid and thorough curing, and subsequent storage in a well-ventilated room at about 33° to 36° F.

(See *4*, *29*, *33*, *41*, *45*, *46*, *52*, *53*.)

SMUT OF ONIONS

(*Urocystis cepulae* C. C. Frost)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Smut occurs in most of the important northern onion-growing sections, including New York, New Jersey, Ohio, Wisconsin, Illinois, Iowa, and Oregon. It apparently does not occur in the southern onion sections even though sets that come from infested areas in the North are used in planting.

Smut is important on onion, leek, and shallot primarily from the point of view of production, because it causes poor stands and stunts the growth of affected plants. The bulbs of affected onions usually are so small that they are unmarketable, although at times they grow large enough to sell. Smutted bulbs shrink excessively in storage. Green onions and onion sets affected with smut are occasionally found on the market.

The disease appears as dark-colored, slightly raised streaks or blisters on the leaves and young bulbs. These pustules are filled with greenish-black, powdery masses of spores. Sets may be greatly shrunken, with the whole exterior covered with blisters. On such specimens, and on others that survive and get into trade channels, the lesions consist of slightly raised brown to black pustules, which are most prevalent near the base of the bulb.

The term "smut" is often incorrectly used by the trade to indicate smudge and black mold rot.

CAUSAL FACTORS

The spores of the causal fungus (*Urocystis cepulae*) are resistant to cold and serve to carry the organism through the winter in the soil. Diseased bulbs and sets are a means by which the fungus is carried over the winter and are also an important source of inoculum when they are set out in the field.

Only very young plants are subject to infection. After the seedlings are about 3 inches high they become immune to further infection. The disease does not develop nor spread in transit or in storage.

CONTROL MEASURES

Because infection proceeds from the soil and from diseased onions, it is advisable not to return infested onion refuse to the soil.

Onions are not susceptible to infection after they are 3 to 4 inches high; consequently, the transplanting of noninfected plants even into smut-infested soil is a practicable measure. Onions grown from sets also are not susceptible and can be planted in smut-infested soils without risk of a smutted crop.

Where the use of sets or seedlings is impracticable, as in the larger onion-growing sections of the North, smut can be controlled by the application of a solution of formaldehyde (1 pint formaldehyde in 16 gallons of water) in the furrow with the seed. The diluted solution should be applied at the rate of 200 gallons per acre, or 1 gallon to about 185 feet of row.

(See *1*, *2*, *39*, *42*, *50*, *51*.)

SUNSCALD OF ONIONS

Onions grown in regions where the temperature is high and the sunlight extremely bright are often severely affected with sunscald. This injury takes place at harvesttime when the bulbs are exposed to heat and bright sunlight. Immature and moist onions are generally injured most severely. The tissues of the exposed area of the onion are killed and become soft and slippery, but soon lose moisture rapidly by evaporation, so that sunken leatherlike areas are produced, which are usually bleached almost white. The scalded areas vary from ½ to 1½ inches in diameter, depending on the exposure and the size of the onion.

Sunscald is often a serious factor in marketing onions, not only on account of the blemishes but because various bacteria and other organisms enter through the dead areas and cause decay during transit and marketing. Bacterial soft rot is usually the most important disease that follows sunscald.

(See *27*.)

WHITE ROT OF ONIONS

(*Sclerotium cepivorum* Berk.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

White rot has long been known in the British Isles, Holland, and France, where it attacks onions and leeks; in Italy and Spain,

where it attacks garlic; and elsewhere throughout Europe. Until 1923 it had been reported from only two onion districts in the United States, having been found in Oregon in 1918 and in Virginia in 1923. Since that time it has caused more or less trouble in California,[4] Virginia,[5] Kentucky, and New Jersey. The disease is of importance in the growing crop as well as after harvest.

The first symptoms of the disease in the growing plant are yellowing and dying back of the leaves, the rate of progress depending upon environmental factors. The affected plant, especially if young, may wilt and collapse completely, or there may be a gradual decline which extends through several days or weeks. If a diseased plant is pulled it will be found that the roots and scale bases are decaying. The affected roots generally die and the scales become semiwatery. The symptoms displayed by the bulb often are like those of other bulb rots, such as gray mold rot and fusarium bulb rot. However, the mycelium is more white and cottony and the sclerotia are much smaller (one thirty-second of an inch in diameter) and more spherical than those of the *Botrytis* species that cause bulb rot (pl. 5, *F*). In fusarium bulb rot no black sclerotia are produced.

CAUSAL FACTORS

The pathogen, *Sclerotium cepivorum*, lives over winter in the soil, from which it attacks the new crop. It can also live over in bulbs and thus be carried into noninfested fields. It is readily carried from field to field by infected bulbs, infected seedlings, and by contaminated containers.

Field observations and laboratory experiments indicate that the disease is most destructive in a moderately cool soil (50° to 68° F.) having a medium moisture content. Such conditions prevail in the onion sections of the Southern States. Whether the climatic conditions in the North are such as to prevent establishment of the disease remains to be discovered. Temperatures below 41° and above 84° prevent the growth of the causal organism.

No data are yet available regarding the development and spread of this disease in transit and storage.

CONTROL MEASURES

The sclerotia of the white rot fungus can remain alive in the soil for several years; therefore fields known to be infested should be avoided for planting onions.

Shallots and leeks are said to be more resistant than onions. (See *12*, *47*.)

PULSE FAMILY

The pulse family (Leguminosae) contains a large number of cultivated plants many of which are important vegetable and forage crops. In this group there are several genera and species of beans and peas that produce fruit pods and seeds which are classed as

[4] GARDNER, M. W. SCLEROTIUM CEPIVORUM FOUND ON GARLIC IN CALIFORNIA. U. S. Bur. Plant Indus., Plant Dis. Rptr. 23 : 36. 1939. [Mimeographed.]

[5] U. S. BUREAU OF PLANT INDUSTRY. WHITE ROT OF ONIONS IN VIRGINIA. U. S. Bur. Plant. Indus., Plant Dis. Rptr. 14 : 104–105. 1930. [Mimeographed.]

vegetables on the market. Among these the common bean, lima bean, and garden pea are of greatest importance.

Beans

The pods or entire fruits of the wax and green pod varieties of the common bean (*Phaseolus vulgaris* L.) generally are marketed for table and canning purposes, whereas the lima bean (*P. lunatus* L., var. *macrocarpus* Benth.) is sold either in pods or shelled. Marketability of the snap bean varieties is determined primarily by tenderness, crispness, good color, and freedom from blemishes and decay; in the case of lima beans filling the pods is of primary importance.

Toughness and poor color may be due to old age. The pods take on a poor color and become flabby, tough, and stringy when they become overmature on the plants and the seeds become large and hard. Too long an interval between picking and consumption also may lead to poor color, flabbiness, and toughness of the pod.

Lack of crispness of the pods may be due to wilting of immature pods either before or after picking. In the former case, drought or diseases of the plant may be the cause; in the latter, excessive drying in the field, in the packing house, or in transit may be responsible, especially if the beans were picked when very young.

A number of diseases, such as bacterial soft rot, cottony leak, gray mold rot (see Peas, Gray Mold Rot, p. 37), rhizopus rot, russeting, and watery soft rot, only rarely cause loss in the field but are exceedingly important in the later stages of distribution and marketing.

Many diseases that decrease production by killing or weakening the plants also directly affect the pods. The most important of these are: Anthracnose, bacterial blight, bacterial wilt, downy mildew, pod blight, powdery mildew (see Peas, Powdery Mildew, p. 39), sclerotium rot, soil rot, and rust. The plants and pods are also affected with heat injury, hopperburn, and sunscald.

There are several bean diseases that seriously curtail production by attacking the plant but do not affect the pods directly. Wilting, toughness, and underdevelopment of pods may be indirect effects of fusarium root rot (*Fusarium solani* (Mart.) var. *martii* (App. et Wr.) Wr. f. 3 Snyder and other species), of mosaic, of leaf spots (*Cercospora canescens* Ell. and Mart., *Cercospora cruenta* Sacc., and *Phyllosticta phaseolina* Sacc.), of ozonium root rot (*Phymatotrichum omnivorum* (Shear) Dug.), of ashy stem blight (*Macrophomina phaseoli* (Maubl.) Ashby), of root knot (*Heterodera marioni* (Cornu) Goodey), or of root rot (*Thielaviopsis basicola* (Berk.) Ferraris).

(See *59*, *60*, *61*, *62*, *63*, *70*, *73*, *76*, *81*, *82*, *86*, *107*, *108*, *114*, *119*, *123*, *132*, *133*, *136*, *139*, *140*, *157*, *158*.)

ANGULAR LEAF SPOT

(*Isariopsis griseola* Sacc.)

This peculiar leaf spot is found on snap beans and has also been reported on peas. Sometimes the pods are attacked, thus making the disease a direct market factor. In cases of severe spotting of the

leaves the vitality of the plants may be so reduced that few or no marketable pods are produced.

On the leaves small angular brown spots closely crowded together are produced; on the pods the spots vary greatly in size and are characterized by two distinct zones of color, the reddish-brown center of each spot being sharply separated from an outer black zone, which in turn is clearly separated from the healthy portions of the pod. Black bundles of spore-bearing mycelium often cover the surfaces of the lesions, giving a characteristic appearance to the larger spots.

No means of control are known.

(See *93.*)

ANTHRACNOSE

(*Colletotrichum lindemuthianum* (Sacc. and Magn.) Briosi and Cav.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Anthracnose occurs in varying degrees on varieties of wax, green pod, pole, navy, and kidney beans, and on some varieties of other species, such as the scarlet runner and the lima bean.[6]

The disease is world-wide in its distribution. Whether or not it will occur in a given locality or during a given season is determined primarily by climatic conditions. Moderately cool, humid or rainy weather during the early part of the growing season is essential for the development of anthracnose in serious form.

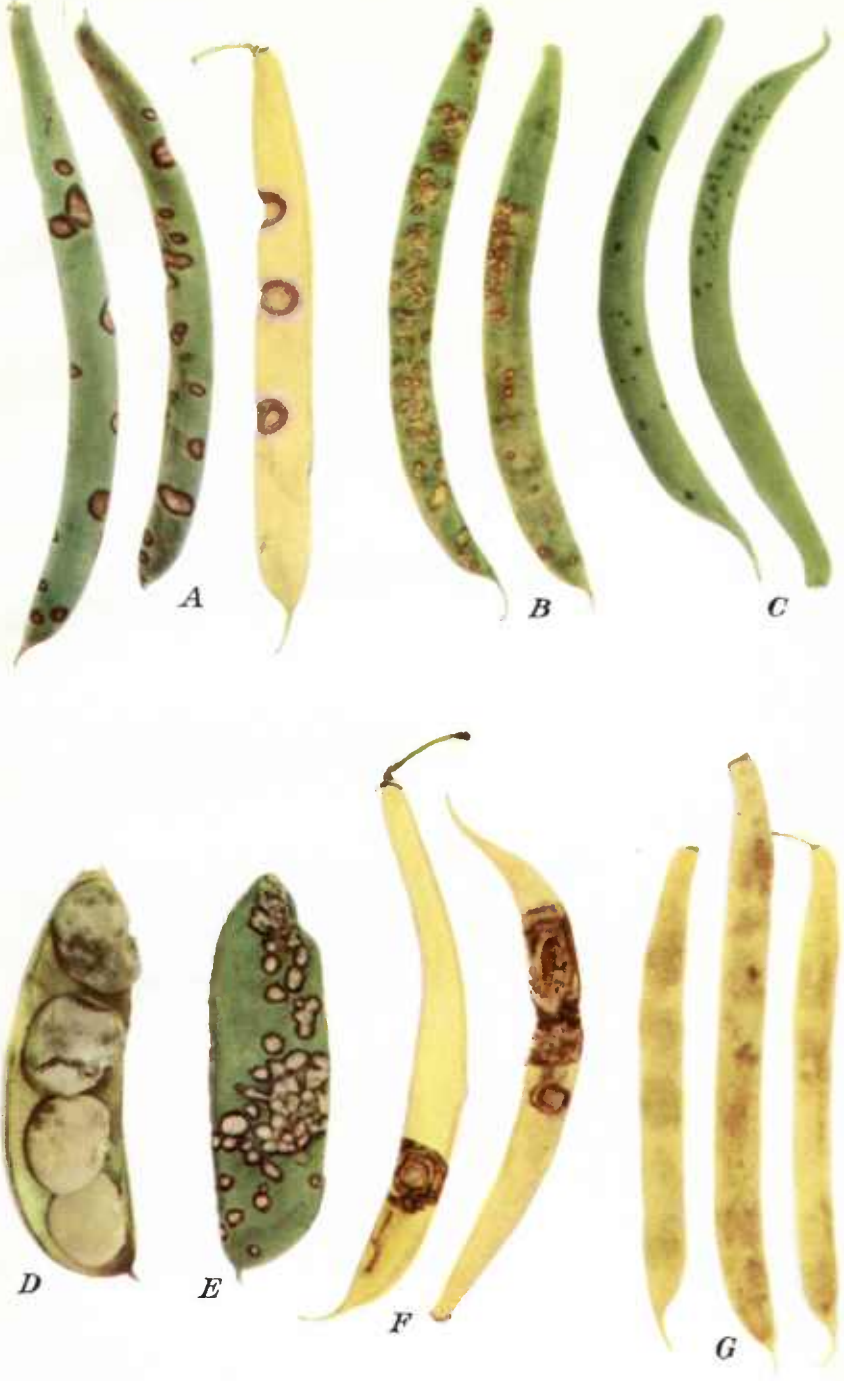
From the point of view of production, the losses are due to poor germination of affected seed, to destruction of diseased seedlings, and to low yields of affected plants. In marketing, the losses are due to spotting of the pods, which renders them unsightly and unsalable for table and canning purposes. The spotting may appear on the pods before or after picking and frequently develops in transit on pods that appeared healthy when packed. Sometimes the fungus penetrates the pod to the seed and causes a spotting, which lowers the selling price of the dry seed.

On the pods, the first symptoms are minute oval or circular greenish-brown specks, which later become brick or rust red to black around the border as they enlarge (pl. 7, *A*). These spots may range in size from mere specks to areas one-half inch in diameter. As the spots enlarge their centers sink and become dark. Under moist conditions, the center becomes covered with flesh-colored dots, which are heaps of fungus spores, and these sometimes run together forming a slimy layer. Under dry conditions, the spore masses dry down to gray, brown, or even black granulations, or to small pimples.

In the case of early infections, the fungus often grows from the pod wall into the seed. When the attack is light, the seed shows only yellowish spots, but when severe, the spots may have yellow, brown, or even black centers with tawny-brown or tan-colored borders. The lesions may occur anywhere on the seed.

---

[6] An anthracnose caused by *Colletotrichum truncatum* (Schw.) Andrus and Moore injures the lima and garden beans to some extent in the Southeastern States. Small, red superficial blotches are the first symptom on the pods. Later the blotches may spread and become brown or grayish and show numerous black fruiting bodies that look like pycnidia. This type of anthracnose apparently is not yet a market factor.



A, Bean anthracnose. *B*, Bean bacterial blight. *C*, Bean bacterial "halo" blight. *D*, Lima bean soil rot. *E*, Lima bean scab. *F*, Bean soil rot. *G*, Bean russeting.

The stem of a seedling may become infected at any point throughout its length. The new lesions at first appear as brown specks in the epidermis; later the spots enlarge lengthwise along the stem and involve the tissues beneath the epidermis, finally causing pits that may be deep and numerous enough to cause collapse of the seedling.

When the leaves are involved, the lesions usually start on the under side of the veins and may lead to death of the leaf tissues around the affected spots. These dry brown areas generally are small, irregular, and elongated. When they are torn by the wind, the leaves become ragged in appearance. Severe attack on the petiole may cause dropping of the leaf blade.

CAUSAL FACTORS

The causal fungus, *Colletotrichum lindemuthianum*, may remain viable through at least 2 years in seed harvested from diseased pods, or may overwinter in the field on trash from diseased plants of the previous season. The free spores when worked into the soil are not viable after 8 to 10 weeks. The introduction of the disease into a field occurs most frequently through the use of diseased seed. Under favorable moisture and temperature conditions the fungus, which has been lying dormant in the seed or soil, begins to grow and develop spores. If the seedling is not killed before it emerges from the ground, it may carry either the old or new spore-bearing lesions above ground. Spores from either source can lead to new infections when moisture conditions are favorable, so long as the tissues of the pods have not passed the brittle or snap stage. For this reason healthy-appearing pods picked during or immediately following rains in diseased fields, or pods from healthy fields when packed with those coming from diseased fields frequently develop spotting in transit.

Growth of the fungus is possible throughout a temperature range from 32° to 93° F., but most rapid development takes place at about 73°. Spores are produced abundantly on bean pods at 50° in 6 days, but only sparingly at 82° and above. On moist, favorable bean tissue spores can produce infections between 57° and 80°, the greatest number being produced at about 71° to 73°.

An abundance of moisture is essential for development and dissemination of the spores as well as for their germination and for infection of the plant. Moisture may be present either as a film on the plant surface or as vapor in the air. Humidities of about 95 percent at temperatures between 64° to 68° F. are favorable for germination of the spores and penetration of the host.

The incubation period ranges from 4½ to 9 days, varying with the temperature, the host, and the age of the tissues. Consequently, pods that look sound when picked and packed may actually be infected and develop lesions during transit and marketing. Young succulent tissues are most easily infected. Pods are not subject to infection after they pass the brittle or snap stage.

Although no snap bean varieties are known to be immune to attack by the fungus, there are all degrees of resistance. At one extreme are those varieties on which only slight lesions are produced on the youngest tissues; at the other are those on which the lesions are

so numerous or severe that the plants wilt and collapse in 5 to 6 days after inoculation. These phenomena are the basis for experimental work in the development of resistant strains by selection and breeding for the control of anthracnose.

CONTROL MEASURES

The use of disease-free seed is a most satisfactory means of control of anthracnose. In some localities hand selection of seeds from pods or from plantings free of anthracnose may be possible. The seed so selected will produce a disease-free crop if it is planted in clean soil. It is usually possible and highly desirable to procure seed grown in sections where climatic factors are not favorable for anthracnose, such as Colorado, Idaho, and California, where a combination of high temperatures and semiaridity or aridity exists.

The use of resistant varieties to control anthracnose is complicated by the fact that there are several forms of the causal organism, and plants resistant to one form may not be resistant to another. The growers in each locality should test suitable varieties for degree of resistance under their conditions.

Precooling and good refrigeration (38° to 44° F.) in transit will check the development of anthracnose infections already present in beans at the shipping point and will prevent the development of new infections for approximately 2 weeks.

(See *61*, *63*, *64*, *72*, *81*, *84*, *100*, *116*, *117*, *118*, *134*, *137*, *152*.)

BACTERIAL BLIGHT AND HALO BLIGHT

(*Phytomonas phaseoli* (E. F. Smith) Bergey et al., *P. medicaginis* var. *phaseolicola* Burk.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Bacterial blights of beans are much more common and destructive than was originally thought. They are now recognized as being very serious in sections where weather conditions are favorable for their development and spread.

One or more forms of bacterial blight are found in practically all important bean-producing sections of the United States and of Europe. This type of disease affects garden, field, and lima beans of many varieties, none apparently being entirely immune, although some seem to show resistance. In addition to seriously interfering with germination of the seed, bacterial blights hinder the growth and development of the plant. A great reduction in yield may be expected in fields of beans that are badly infected, and those pods that do mature may become so spotted that they are worthless by the time they reach the market. Small spots that are easily overlooked at shipping point may enlarge during transit and either make the product worthless or reduce its market value to such an extent as to make the shipping of affected beans extremely unprofitable.

On the pods the bacterial blight is indicated by small, watery spots that gradually enlarge, forming irregular blotches varying in color from amber yellow to almost brick red around the margin (pl. 7, *B*). The bacteria often ooze out of the center of the spots and show as

a greasy yellowish slime. This finally dries, forming a yellowish-brown crust on the surface. In severe cases the bacteria penetrate the pod and attack the seeds. Lesions on the seed usually are small, irregular, yellow spots or blotches; in severe infection the whole seed may become shriveled and yellow.

In the field, bacterial blight on the leaves is first indicated by small, irregular, water-soaked areas, bordered by reddish lines. Later, large areas of the leaves may become involved, finally drying out and becoming brown and brittle with age. Bacteria washed down from the cotyledons or leaves sometimes produce reddish-brown lesions on the stem of the plant, or produce water-soaked areas that enlarge and girdle the stem at the nodes. Plants thus affected often break over at the diseased nodes. In continued muggy weather badly affected plants may have the appearance of being scalded; the pods do not fill out and the ripening is uneven.

CAUSAL FACTORS

The pathogen of the common form of bacterial blight, *Phytomonas phaseoli*, is carried over from year to year in or on diseased or contaminated seed, or in bean trash that remains in the soil. The growing seedlings bring the bacteria above ground in the developing lesions. During damp warm weather the bacterial masses ooze out of these tissues, thus making possible the spread of the bacteria by insects, water, and other contact agencies. The bacteria are very resistant to sunlight and drying. Consequently they are readily disseminated by the wind in fragments of leaf lesions and with soil particles and can lie dormant until proper moisture conditions favor their growth and entrance into plants through the stomata.

Pods that develop the disease in transit do not necessarily show symptoms at time of harvesting or loading. Incipient lesions may develop in transit, but there is no evidence of any new infection after harvest.

A form of bacterial blight usually spoken of as halo blight, caused by *Phytomonas medicaginis* var. *phaseolicola*, is becoming increasingly important in many bean-growing regions. This disease often is more serious and causes more wilting of plants than the common form. Many symptoms are practically identical in the two diseases, but usually the halo blight can be distinguished by the grayish-white bacterial exudate on the leaf and pod lesions and the characteristic light or pale-green halo surrounding the lesions. On the market the pod spots generally show more water soaking, are usually more circular in outline, and they remain greenish gray instead of taking on the yellow and reddish-brown shades typical of the common bacterial spot (pl. 7, *C*).

CONTROL MEASURES

Crop rotation and the planting of disease-free seed are the usual recommendations for the control of bacterial blights. However, by selection and breeding some fairly resistant varieties have been obtained. Although no varieties show absolute immunity, some of the Refugee types exhibit a high degree of resistance. Unfortunately,

some varieties that show resistance to the common bacterial blight are not resistant to the halo type of blight or to bacterial wilt (*Phytomonas flaccumfaciens* (Hedges) Bergey et al.).

The common bacterial blight organism may cause decay of beans throughout a wide temperature range (34° to 95° F.), but development is slow at 45° and below.

Neither spraying of plants nor seed treatment with chemicals or with heat has given satisfactory control.

(See *71*, *74*, *75*, *76*, *77*, *81*, *85*, *92*, *103*, *130*, *131*, *135*, *155*, *156*.)

BACTERIAL SOFT ROT

(*Erwinia carotovora* (Jones) Holland)

Snap beans under transit conditions are less frequently affected with bacterial soft rot than are most other vegetables. However, this decay often follows bacterial blight and frequently occurs simultaneously with some of the fungus rots.

The pods become affected with bacterial soft rot only when they are wet and warm. The causal organism does not enter through the unbroken epidermis, a fact which probably accounts for the relative unimportance of bacterial soft rot on the market, as beans are less subject to breaks and bruises during harvesting and marketing than most vegetables.

For a complete discussion of this disease see Celery, Bacterial Soft Rot, page 46.

BACTERIAL SPOT

(*Phytomonas vignae* (Gardner and Kendrick) Bergey et al.)

All varieties of lima beans tested have been found susceptible to bacterial spot. Although the disease sometimes seriously affects the vines and pods, so far it has not been an important factor on the market.

Small, brown spots surrounded by a water-soaked halo characterize this disease on the pods. A straw-colored crust of exudate may be apparent on the surface of some lesions. Spots may occur on any part of the pod, and frequently they penetrate the walls, causing a reddish discoloration of the seeds. Spongy excrescences are sometimes found protruding from the inner walls of the pod beneath the surface lesions.

Bacterial spot on the leaves is characterized by brownish- or purplish-colored, irregular lesions. At first the spots appear as small brown points on the upper surfaces of the leaves, but they are never water-soaked, like those characteristic of most other bacterial diseases. The centers of the large lesions turn gray or straw-colored, whereas the margins remain glistening and purplish. Individual lesions vary from one-sixteenth to one-eighth of an inch in diameter. Spots sometimes coalesce and form large, ragged areas from which some of the dead tissue eventually drops out. Reddish-brown or caramel-colored streaks occur on the veins and petioles, and when the base of the leafstalks is attacked the leaves are shed prematurely.

Lesions on the stem vary greatly in size, some reaching a length of 2 or 3 inches. Occasionally a light wine-colored exudate appears

on the stem lesions, and dries to form a glistening crust. In severe attacks the petioles are killed, which causes the blossoms and young pods to drop.

The pathogen, *Phytomonas vignae*, enters the plant through the stomata and extends downward into the tissues, causing death of the cells. Frequent rains and temperatures between 80° and 86° F. are most favorable for the dissemination of the organism and the development of the disease.

Since it is thought that the bacteria are carried over winter in infected seed, the selection of disease-free seed appears to be the most promising method of control.

(See *65*, *90*, *91*, *147*.)

BACTERIAL WILT

(*Phytomonas flaccumfaciens* (Hedges) Bergey et al.)

Bacterial wilt in some respects is so similar to bacterial blight that it has been confused with the latter in many bean-growing regions. The pathogen has been found in bean seed from many States, but generally it does not cause as much loss as the blight organisms. Green beans on the market do not often show this disease.

As the name implies, the disease is primarily a wilt. Infection proceeds systemically through the vascular system of the plant into the pods and seed. At times the pods bearing infected seed seem perfectly healthy; at others, the infection follows the seed-bearing suture causing dark-green water-soaked elongated areas. In immature pods the diseased areas may be yellowish green and somewhat withered. The lesions are more conspicuous on mature pods, where they appear as greenish-brown areas in the normal yellow of the pod. No pod spots of the blight type are produced.

On the seeds the lesions are essentially like those caused by *Phytomonas phaseoli*, with a marked tendency toward yellow discoloration at the point of attachment of the seed.

The causal organism lives in bean seed and can retain its virulence for at least 5 years. When infected seed is planted and conditions favor growth of the organism, it spreads into the vascular tissue of the growing plant, causing wilt. By way of the vascular tissues it proceeds into the pod and on through the hilum into the seed.

Control is most easily effected by planting seed selected from wilt-free fields.

(See *71*, *102*, *103*, *119*, *156*.)

COTTONY LEAK (WILT)

(*Pythium butleri* Subr.)

Cottony leak is sometimes a market factor in the shipment of snap beans from Florida and other Southern States. During rainy weather the pods become infected, apparently by contact with the causal fungus in the soil, and large water-soaked lesions are produced, which often enlarge greatly during the transit and marketing period. If beans are harvested and packed while wet, the causal organism frequently grows profusely over the surface of the beans, matting the

pods together into nests. The abundant white cottony mycelium and the leaky condition of the pods have given rise to the common name "cottony leak" to describe this disease on the market. Cottony leak appears very similar in many respects to watery soft rot on beans. However, the cottony leak organism usually produces a greater abundance of white cottony mycelium, but it never produces the black sclerotia that sometimes characterize the advanced stages of watery soft rot. Furthermore, it grows more rapidly at high temperatures, whereas the watery soft rot organism develops best at cool to moderate temperatures.

Although species of *Pythium* have long been known to cause root rots and damping-off of seedlings, apparently it was not until 1930 that a serious wilt of beans caused by this type of organism was reported and then from localities as widely separated as Colorado and Virginia. It was found that the organism infects the stems of the bean plants at the soil line and causes a water-soaking of the tissues, which often progresses upward into the branches and lower leaves. The affected tissues are not discolored but are water-soaked and softened so that they slip off of the more woody vascular tissues. If the temperature and moisture conditions are favorable, some plants are wilted within a few days after infection takes place. At temperatures near 90° to 100° F. the wilt progresses very rapidly. Plants severely affected rarely produce any marketable pods.

All varieties of snap beans appear to be susceptible to the cottony leak organism. No control measures are known except to discard at the packing shed all beans that show water-soaked lesions. Beans harvested from fields known to be infested should be surface-dried and cooled to a temperature of 50° F. or below as soon as possible, in order to prevent the decay from spreading from one pod to another during transit.

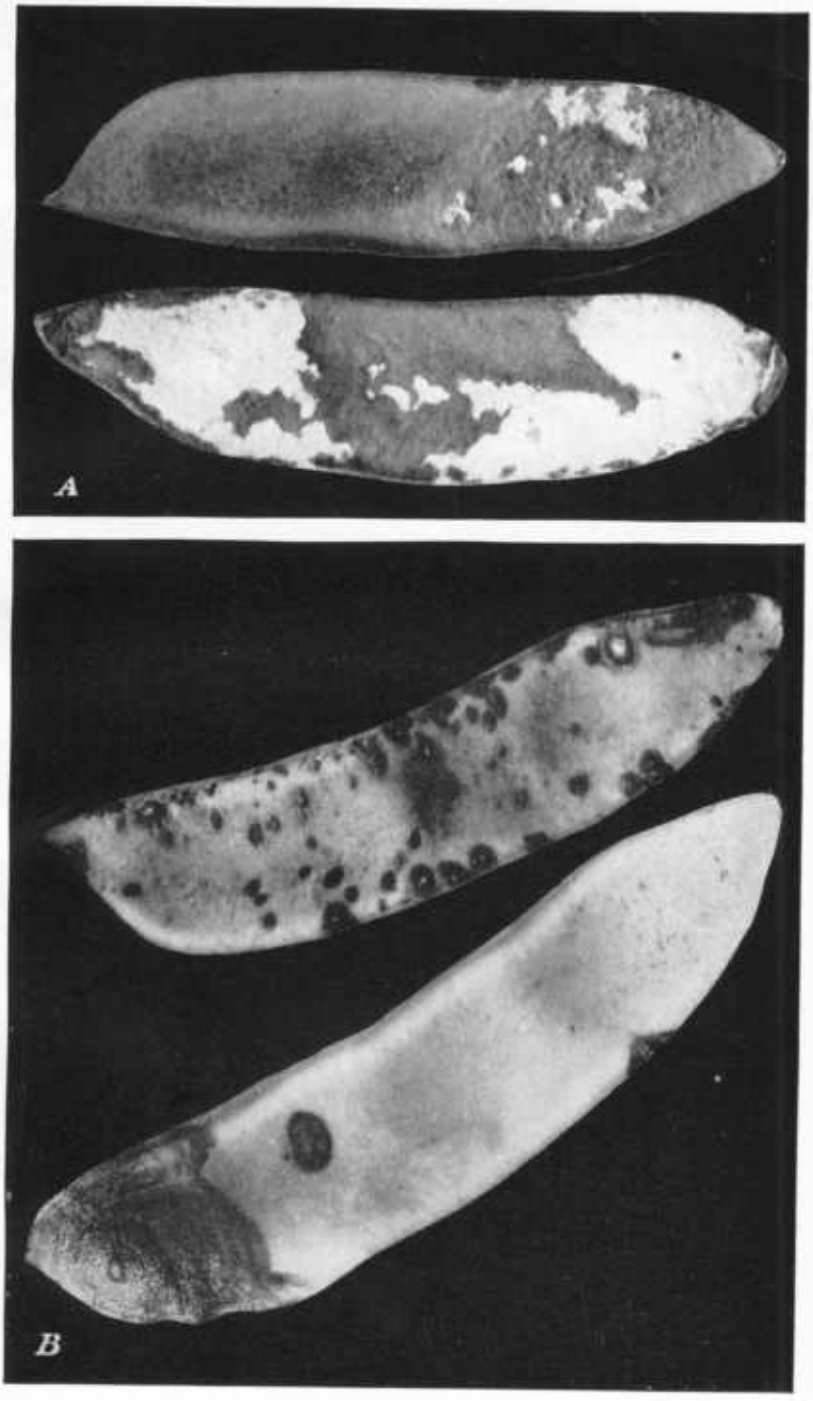
(See *98*, *99*.)

DOWNY MILDEW

(*Phytophthora phaseoli* Thax.)

Downy mildew is one of the important diseases of lima beans, affecting both the bush and pole varieties. It was first described in 1889, and since that time has been reported as causing more or less serious damage each year. Downy mildew is prevalent mostly in the Northeastern States. Reports from Connecticut, New York, Delaware, New Jersey, Pennsylvania, Virginia, West Virginia, and Ohio have cited losses due to this disease during recent years.

The fungus is most often observed on the pods, but it may also attack other parts of the plant. It appears as white, woolly masses, consisting of dense patches of mycelium and conidiophores, which usually are first noticed on one side of the pod, and under favorable conditions spread to all parts of the pod (pl. 8, *A*). Injury to the pod usually is limited to the area covered by the fungus. A purplish border sometimes separates the healthy green tissue from the diseased area. The fungus eventually may work through the pod and infect the seeds. Severely affected pods wilt and shrivel. Young pods may be killed outright or injured so that they do not mature. When affected pods do mature their appearance destroys their market value.



A, Lima bean downy mildew. *B*, Lima bean pod blight.

The lesions also open the way for attack by saprophytic or weakly parasitic organisms in transit and in storage.

The pathogen, *Phytophthora phaseoli*, overwinters as dormant mycelium or as oospores in affected seeds or in trash in the field. If moist moderately cool weather prevails the fungus produces spores that are disseminated by insects, wind, and rain. At times, the spread of the fungus is largely accomplished by bees visiting blossoms and carrying the spores from one plant to another.

For control of this disease the use of clean seed is recommended, and also the burning of bean trash, which may harbor the fungus over winter. Spraying with bordeaux mixture may be advisable in some localities along the Atlantic coast.

(See *79*, *94*, *100*.)

POD BLIGHT

(*Diaporthe phaseolorum* (Cke. and Ell.) Sacc.)

Along the Atlantic seaboard pod blight of the lima bean occasionally causes lesions on the pods and may penetrate to the seeds, rendering both unsightly. Pod blight generally does not become severe until late in June or early August, when the vines are nearly full-grown. Young pods rarely show lesions.

In the early stages the lesions consist of circular or oval spots of darkened tissues; later, they become studded with minute gray elevations that soon break the skin of the pod and emerge as black pustules or fruiting bodies (pycnidia) (pl. 8, *B*). In advanced stages the entire pod may become diseased and covered with pycnidia.

The causal fungus overwinters in seeds or in bean trash. Under warm moist conditions it produces pycnidia, from which the spores are exuded during wet weather to be later disseminated by wind, water, or other means. The leaves of the plant are attacked first and usually have a good growth of pycnidia available by the time the pods become old enough to be susceptible. Young pods are not attacked. The fungus enters readily through wounds and the first symptoms of disease appear about 4 days after inoculation.

Because the pathogen apparently overwinters and is carried into the field on seed, it is recommended that only disease-free seeds be planted.

(See *95*.)

RHIZOPUS SOFT ROT

(*Rhizopus tritici* Saito, and *R. nigricans* Ehrenb, ex Fr.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Under favorable conditions for infection practically all kinds of vegetables are subject to rhizopus soft rot. It occurs in vegetables from all regions, and although it is rarely of any significance in the field, during transit and marketing it often causes very serious losses. It is much more important on beans than peas but sometimes occurs on the latter. All varieties of pod and shelled beans may suffer from this decay if they are moist in transit and if moderately high temperatures prevail.

In active stages of development rhizopus rot is always more or less soft and watery. In many instances enough water is liberated to cause a leaky condition, but in very dry atmosphere sometimes the water is lost rapidly enough by evaporation to cause shriveled, moderately dry areas. Under moist conditions there is produced on the surface of lesions a coarse, white, stringy mycelium, which bears glistening, white, spherical heads (sporangia) that soon turn black and break open, releasing thousands of dustlike black spores. This bushy growth of mycelium often extends an inch or more from the surface and is sometimes called "whiskers." Eventually this mold collapses and becomes matted and black from the liberated spores. These characteristics of the fungus usually serve to identify the decay.

The fungus spreads readily by contact between diseased and healthy beans or peas in the center of moist hampers during transit. The masses of pods surrounded and held together by this mold are known as "nests" (pl. 9).

CAUSAL FACTORS

Various species of *Rhizopus* are ever present in the air, soil, and water in all regions; consequently, most products are exposed to inoculation at some time during the harvesting, packing, and shipping period. Wounds or bruises and surface moisture on beans, peas, and other products greatly favor infection by *Rhizopus*. The later development of decay depends largely upon temperature conditions. Snap or green beans have been found showing rhizopus rot throughout a range of temperatures from 54° to 96° F. with most active development taking place near 80° to 85°. The temperature range varies among different species of *Rhizopus*, but generally speaking rhizopus rot is not a serious factor in cars of produce unless temperatures therein are well above 50°.

CONTROL MEASURES

It is recommended that insofar as possible all excessively wounded and bruised stock and that showing any sign of disease at time of packing be discarded. Beans and peas should be cooled and surface-dried as soon as possible after picking, and shipped under refrigeration sufficient to keep the warmest part of the load below 50° F.

(See *117*.)

RUSSETING

Russeting is a surface discoloration of snap bean pods that develops primarily after harvest. It also occurs in the field to some extent on both green pod and wax pod varieties but is much more common in the latter. It is very objectionable from the market standpoint because of the appearance of the stock.

The lesions consist of chestnut-brown surface discolorations of various shapes and sizes, sometimes involving the entire pod (pl. 7, *G*). At times discolored streaks are more or less parallel, like those of sunscald, but they differ from these in that they are not as red and occur on both sides of the pods. The brownish discolorations result from the death of three or four outer layers of cells. The dead tissues are not sunken, and they do not decay unless invaded by bacteria or fungi.

Bean rhizopus rot.

Nothing is known regarding the cause of the injury. No organisms are involved. It may be a matter of the death of cells that for some reason are more sensitive to external factors than their immediate neighbors or that have a higher metabolic rate and consequently break down more rapidly. Moist, warm beans held in a confined space have been observed to develop somewhat similar discolorations thus suggesting that russeting may be due in part to respiratory trouble. Carloads of green beans have been known to develop serious russeting during long transit periods.

A somewhat similar type of surface discoloration of bean pods is sometimes associated with mosaic.

RUST

(*Uromyces phaseoli typica* Arthur)

Bean rust is world-wide in distribution and to some extent is found each year in most bean-growing localities of the United States. It may be very serious in localized areas, sometimes causing total loss of the crop, but usually it is of minor importance in the field as well as on the market.

There is a considerable range in susceptibility to rust among varieties of field and garden beans. Some of the pole beans and dwarf field beans may be very severely affected. Dwarf garden varieties of beans are sometimes affected late in the season under cool, moist conditions, but ordinarily only slight loss is sustained.

Rust causes most severe injury to the leaves of the bean plant, although the stems and sometimes the pods are affected. The first symptoms of infection are small dark-green or brownish raised pustules commonly found on the under side of the leaves. These pustules soon turn reddish brown and break open releasing innumerable spores as a rustlike powder. Later in the season black powdery spore masses are produced in pustules similar to the rust-colored ones. In severe infections the leaves may die or the green leaf tissue may be so reduced that the plant is not able to manufacture food enough to set and develop pods.

When bean pods become infected with rust small brown spots are produced similar to those formed on the leaves. In advanced stages these spots appear as rusty-brown pustules, which vary from one-sixteenth to one-eighth inch in diameter and show definite slits and feathering of the broken epidermis over the rusty spores within. Although these pustules do not develop into decay they sometimes blemish the stock sufficiently to reduce its market value.

The causal fungus, *Uromyces phaseoli typica*, overwinters in bean trash, and this serves as a source of inoculum the next season.

The minimum temperature for spore (uredospore) germination is 35° F., the optimum 58°, and the maximum 94°. The optimum for infection is about 63°. Regardless of temperature conditions, no infections occur unless the relative humidity is very high (95 percent). A period of high humidity lasting for at least 8 hours is necessary for infections to take place.

Varietal resistance to bean rust is so marked and has been so well demonstrated in various localities that an effective control is now within reach of all bean growers. The choice of varieties

depends upon local conditions; therefore local authorities should be consulted.

(See *87*, *88*, *97*, *100*.)

SCAB

(*Elsinoë canavaliae Rac.*)

Although scab has not yet been found on beans grown in the United States, it is known to occur in Central America and Mexico and is one of the common and most destructive diseases of lima beans in Cuba and Puerto Rico. Over a period of years the lima beans imported from these localities have shown more or less spotting by this disease.

On the pods conspicuous red, brown, and maroon, swollen, oval lessons varying in size from one thirty-second to one-half inch in diameter are characteristic symptoms (pl. 7, *E*). The spots are scattered irregularly over the pod, but they do not penetrate to the seeds. However, badly spotted pods are injured in appearance to such an extent that they are unmarketable, even though the seeds within are healthy.

(See *68*, *69*, *104*, *105*.)

SCLEROTIUM ROT (SOUTHERN BLIGHT)

(*Sclerotium rolfsii* Sacc.)

As a seedling blight and stem decay of bean plants sclerotium rot sometimes causes serious damage, but on the marketable pods of snap beans it seldom is important. Under wet soil conditions in the Southern States, bean pods that touch the soil or become spattered with mud during rain may contract the disease. The decay produced is watery, but not much change in color takes place in the affected tissues. The best diagnostic symptom is the white, silky fanlike mycelium growing over the surface of the pods, and in advanced stages the presence of brown spherical (mustard-seed-like) sclerotia.

Sclerotium rot is also of minor importance as a semiwatery decay of carrots and garlic. This decay of these products has been observed most often on the Chicago market in shipments from Louisiana.

Inoculation experiments with bean pods indicate that infection will not take place at a temperature below 46° F. Consequently temperatures of 40° to 45° in transit will check the extension of old lesions and prevent the formation of new ones.

(See *117*.)

SEED SPOTTING

(*Cladosporium herbarum* (Pers.) Link)

Shelled green lima beans frequently show an undesirable spotting that greatly reduces their value by the time they reach the market. Most of this spotting is caused by a species of *Cladosporium* that inhabits decaying vegetable matter and often blemishes the pods of beans and peas. During the shelling process the seeds are con-

taminated with this fungus and spotting results if moisture and temperature conditions favor its development. Usually, small brown spots one-sixteenth to one-eighth inch in diameter are produced on the seed coat, but under humid conditions they may become larger and show an olivaceous color due to a superficial growth of the causal fungus. In advanced stages of decay greenish-brown granular spore masses are formed over the larger spots and on removal of the seed coat brown spots may be found on the seed (cotyledons).

Experiments have shown that spotting may be controlled for a week by a temperature of 40° F.; at 32° beans are safe from spotting for about 2 weeks. Where it is feasible to use solid carbon dioxide (dry ice) as a refrigerant the increased carbon dioxide content of the air assists greatly in retarding development of the causal fungus. Beans held in an atmosphere containing 25 percent or more of carbon dioxide will develop little or no spotting, and the flavor and texture of the beans are not injured.

Tests of various washing solutions have shown that a 4-percent solution of chlorinated lime will give excellent control of seed spotting if the pods are washed in this solution before the seeds are shelled.

(See *67*.)

SEED STICKINESS

Although many kinds of bacteria may cause a sticky condition of the surfaces of shelled green lima beans under transit and storage conditions, it appears that *Pseudomonas ovalis* Chester, *Achromobacter coadunatum* (Wright) Bergey et al, and an organism similar to *A. lipolyticum* (Huss) Bergey et al. are the most common causes of this trouble. These soil organisms contaminate the seeds during the shelling process. When temperature and moisture conditions favor their development on the seed coat a sticky or slimy condition results.

Prompt refrigeration is an effective control measure for the prevention of stickiness. A temperature of 32° F. will prevent the development of stickiness for 2 weeks; at 40° good control may be expected for about a week.

Stickiness may also be prevented by washing the shelled beans for 1 minute in a 30-percent solution of ethyl alcohol and allowing them to dry for a short time before packing them for shipment or storage.

Washing the pods in a 4-percent solution of chlorinated lime before shelling has been found to give good commercial control of stickiness.

(See *67*.)

SOIL ROT (RHIZOCTONIA STEM AND POD ROT)

(*Rhizoctonia solani* Kühn; *Corticium vagum* Berk. and Curt.)

Soil rot affects the root, stem, and pod of the bean. In the field it causes damping-off, stem rot, and pod rot; in transit it often causes severe decay of pods. The fungus spreads readily by contact under moist conditions and is one of the organisms responsible for the "nesting" of beans. This term merely implies that within a hamper or other package there is a mass of decaying snap beans held together by a copious growth of mycelium.

Soil rot is of commercial significance in long-distance shipments, especially those sent from the Gulf States during the earlier months of the year, when wet, soil-stained pods are packed.

Irregular, reddish-brown lesions are produced on the roots and stems of beans in the field. The pods are affected both in the field and in transit. In the field those in contact with the soil usually become infected at the end of the pod. The lesions are large, often irregular in shape, and light brown with soft, badly rotted underlying tissues (pl. 7, *F*). In advanced stages the seeds are discolored (pl. 7, *D*). The larger lesions are often concentrically marked with alternating light- and dark-brown zones and frequently become covered with sclerotia.

The presence of the fungus itself is the best diagnostic character. The mycelium is white at first, but later deepens in color, passing through yellow brown to deep chocolate brown. Most characteristic, however, are the sclerotia or resting bodies. These appear first as small, soft, cream-colored masses, later becoming large and irregular and turning dark brown, similar to the sclerotia formed by this fungus on potatoes, carrots, and other vegetables.

Inoculation experiments with green bean pods have shown that infection may take place in 5 days at 51° F., but the rate of development of decay is very slow below 60°. Most rapid decay occurs in green beans when the temperatures are between 75° and 90°.

Although the causal organism is usually present in the soil, it does not cause serious injury to growing plants unless the soil is wet and the temperature is between 55° and 80° F.

No really satisfactory method of controlling this disease in the field is known. Green beans showing brown discoloration or water soaking should be discarded before the crop is packed for shipment, and sufficient refrigeration should be provided to maintain a temperature of 50° F. or below during transit.

(See *62*, *117*, *140*.)

SUNSCALD

The symptoms of sunscald and russeting at times are very similar but with this difference: Sunscald occurs only in the field, whereas russeting appears to occur primarily after harvest. Sunscald does not occur so long as the pods are well shaded by the leaves. If the leaves fall, whether because of disease or ripening, sunscald is likely to occur on hot days with intense sunlight. The actinic effect of the sun's rays, together with the high temperature, causes the death of the more sensitive surface cells. The first indications of the disease are very tiny brown or reddish spots upon the exposed surface of the pod. These gradually lengthen until they become streaks which run across the pod diagonally and are more or less parallel. The affected areas are water-soaked and brown at first and may become sunken. Often the individual spots fuse to form brown to reddish areas of varying size, which at times cover most of the surface.

The lesions, especially in the early stages, are not easily distinguished from bacterial spot, but they always lack the greasy exudate that often is present in blight lesions.

Like russeting, sunscald affects only the appearance of the pods. In sunscald the seed coats at times are affected, but there is no loss of keeping quality or of viability.

(See *124*, *125*, *126*.)

WATERY SOFT ROT (SCLEROTINIA ROT)

(*Sclerotinia sclerotiorum* (Lib.) DBy.)

Watery soft rot frequently causes serious losses in green beans and peas that are harvested following wet weather. It is often particularly damaging to southern beans shipped in late winter and early spring. The affected pods show water-soaked green to greenish-tan lesions of various sizes and shapes, and usually a surface growth of white cottony mold. In advanced stages of decay the mold spreads from pod to pod, enmeshing them to produce the so-called "nests" of moldy pods in the hampers during transit. Although low temperatures (40° to 45° F.) retard the development of decay somewhat, they will not entirely control the growth of the fungus. Experiments have shown that this type of decay will develop from ¼ inch to over 1 inch lengthwise of bean pods in less than a month when these are held continuously at 32°. Visible decay has been observed to develop in inoculated green beans in 15 days at 32°, in 11 days at 35°, in 9 days at 42°, in 6 days at 46°, and in 4 days at 54°. For a complete discussion of this disease see Celery, Watery Soft Rot, page 56.

(See *117*, *127*, *128*, *203*.)

YEAST SPOT

(*Nematospora phaseoli* S. A. Wingard)

Yeast spot so far seems to be of rather localized distribution, having been reported on lima bean in only a few States. The disease affects the marketability of the shelled lima beans by rendering them unsightly. It also is responsible for a reduced yield by causing premature death of seeds or by preventing seeds from growing to normal size in the pod.

The seeds alone are affected, consequently pods that appear perfectly healthy may contain badly diseased seeds. The seeds within the pod may be attacked at any stage of development, but greater damage is done if they become infected before they are half-grown. Dark-brown, irregular, sunken areas are produced, but usually the seed coats remain unbroken. The affected tissues are grayish brown and granular in texture. The pathogen is a yeast that grows best at a temperature of about 85° F., and makes very little growth at 65°.

Apparently the organism gets to the seeds through wounds, for inoculation experiments have proved successful only when the pods and seeds were punctured. The first symptoms are evident within 2 or 3 days after inoculation, and prominent lesions are formed within a week.

No control measures have been worked out.

(See *59*, *154*.)

303920°—41——5

PEAS

The common garden pea (*Pisum sativum* L.), a member of the pulse family, is grown extensively for its edible seeds. Usually the pods are not eaten, but in a few varieties the pods are sometimes used for food. Regardless of kind or variety, the marketability of peas depends chiefly upon the pods being fresh, green, well filled, and free from blemishes and decay, as well as upon the tenderness and flavor of the seeds. There are many diseases and growing conditions that have direct and indirect effects upon the market qualities of peas.

The seedling diseases (damping-off), produced by *Pythium* spp., *Rhizoctonia* sp., and *Sclerotinia sclerotiorum* (Lib.) DBy. (see Celery, Watery Soft Rot, p. 56), either kill the plants outright or so reduce their vitality that both the quantity and quality of marketable peas are seriously affected. Similar effects are produced by other fungi which cause root rots and wilt [7] (*Fusarium* spp. and *Aphanomyces euteiches Drechsler*). Of the diseases which directly affect both the vines and the pods, the most important are bacterial spot and pod spot. Several diseases like anthracnose, downy mildew, septoria leaf spot (*Septoria pisi* West.), and powdery mildew cause no great loss unless seasonal conditions happen to be especially favorable for the growth of the organisms. Mosaic frequently affects the foliage so severely that fruit-pod production is greatly diminished, and sometimes the pods are seriously distorted. On the market, bacterial soft rot (p. 46), gray mold rot (p. 37), rhizopus rot, and watery soft rot (p. 56) are the most serious diseases. Scab and mosaic are sometimes serious market defects.

Blemishes caused by insects such as thrips and the serpentine leaf miner (*Agromyza pusilla* Meig.) are occasionally serious marketing factors.

(See *66*, *83*, *96*, *101*, *109*, *110*, *111*, *112*, *120*, *151*, *158*.)

ANTHRACNOSE

(*Colletotrichum pisi* Pat.)

In general, anthracnose of pea appears so similar to pod spot that probably the two diseases often have been confused. During a few seasons the disease has been serious in some localities in Wisconsin. It is quite evident that under suitable conditions it has possibilities of producing great damage in some pea-growing sections of the United States, if the fungus once becomes established. It affects the plants in the field, lowering their productivity, and also spots the pods so that they become unsightly and are unfit for marketing.

There is one report of the appearance of this disease on the market. Although it is possible that some spotted pods have been passed as pod spot, it seems probable that anthracnose will not be of market importance until the disease becomes more widely distributed.

All aerial parts of the plants are affected. On the leaves, small irregular spots are formed with light-brown centers and grayish-brown

[7] LINFORD, MAURICE B. PEA DISEASES IN THE UNITED STATES IN 1928. U. S. Bur. Plant Indus., Plant Dis. Rptr. Sup. 67 : 1–14. 1929. [Mimeographed.] (See p. 10.)

margins. On the pods the spots are more nearly circular in shape, are larger, and have light centers with dark-brown borders. Gold-yellow or salmon-tinted spore masses are produced in the center of the pod lesions and in the elongated stem lesions.

The causal agent, *Colletotrichum pisi*, is a close relative of the fungi that cause anthracnose of beans and of cucurbits. The spores are disseminated primarily by water, insects, and man. At 75° F. the spores can germinate, penetrate the epidermis, and establish the fungus in about 36 hours. It is not known how the fungus overwinters, although it is not unlikely that it remains alive in infected seeds and in plant refuse left in the field.

No control methods have been worked out for this disease. In view of the possibility of the fungus living over in the pea seeds, it is advisable to take precautions to reject all discolored pea seed.

(See *111*, *112*.)

BACTERIAL BLIGHT

(*Phytomonas pisi* (Sack.) Bergey et al.)

Bacterial blight has been reported from practically all pea-growing States. It is especially severe in regions that have high humidity. The disease usually becomes apparent first on the lower part of the plant and may eventually affect all above-ground parts. Early infections of seedlings may result in death; later ones may cause a reduction of the crop; and the market value of the pods is often seriously affected because of the conspicuous spots produced upon them.

Infections may take place through stomata or through wounds. Early stages of infection on pods appear as small water-soaked spots; the more advanced stages usually found during marketing are slightly sunken greasy or water-soaked spots of irregular outline with gray or grayish-brown centers (pl. 10, *B*). Small spots overlooked at packing time and infections starting through wounds during the harvesting and packing process probably account for the high percentage of infected stock sometimes found upon the market. The lesions present at time of shipment may enlarge somewhat during transit, but it is not known whether any new infections can take place. In advanced stages of decay the bacteria may penetrate the pod wall and discolor the seed, but this stage is seldom found on the market. The stem and leaf lesions appear as water-soaked green or greenish-brown areas somewhat similar to those found upon the pods.

The causal organism (*Phytomonas pisi*) is carried over from year to year in and on affected seeds. Experimental evidence indicates that the organism does not live over in the soil. Spreading of the bacteria in the field is thought to be due to spattering rain and to insects.

Pea seed grown in the Western States under dry conditions is not likely to harbor the blight organism. This kind of seed is recommended for planting in regions having climatic conditions favoring the development of bacterial blight. There are indications of considerable varietal variation in susceptibility to this disease; consequently, resistant varieties may be developed eventually by breeding and selection.

(See *101*, *106*, *122*, *141*, *142*.)

DOWNY MILDEW

(*Peronospora viciae* (Berk.) DBy.)

Although downy mildew may occur in practically any part of the country in which peas are grown, it is seldom of economic importance except in the humid regions along the coast of California, Oregon, and Washington. Both field and garden varieties of peas are susceptible to this disease.

The first symptoms of downy mildew appear as small yellowish spots on the upper surface of the leaflets. As these areas enlarge, a whitish-gray or drab-colored mildew may be observed on the under side of affected leaflets. This mildew is made up of the mycelium and numerous spores of the fungus. If severely infected a whole leaflet may shrivel, turn brown, and die. Occasionally, affected leaves show grayish-white porcelaneous areas, which indicate that the fungus is producing resting spores (oospores) within the tissues. Frequently there is also severe infection of the pods, and if these are not discarded before packing for shipment, the lesions are considered a serious blemish on the market.[8] Pod infections show as yellowish slightly raised irregular blotches, but there is no surface mold visible (pl. 10, *C*). The internal tissues of the pod wall in these yellowish blisterlike areas are filled with oospores. These blotches do not become soft or decay unless some other organism invades the tissues. In affected pods the seeds underneath the yellow blotches on the pod wall are usually aborted or undersized and may show discoloration. The inside of the wall of affected pods usually shows white velvety proliferations under the yellow areas (pl. 10, *D*). These white velvety patches have the appearance of a heavy coat of mold, but in most instances no mycelium is visible in them. Other injuries and diseases sometimes stimulate the pod wall to develop similar white patches. (See Scab, p. 39.)

Downy mildew does not cause serious damage to peas, except in cool regions where fogs and rains keep the foliage moist. The spores of the causal fungus are spread by winds and splashing water. They can germinate and cause new infections overnight at temperatures between 50° and 70° F. It has been shown experimentally that some infection may take place even at 32°, but the most active growth is made at near 60°, and little or no development occurs at 80°.

If climatic conditions favor the development of the causal organism, the control of downy mildew in peas is very difficult. It is thought that the fungus is carried over from year to year as perennial mycelium and oospores in decaying vines and leaves that become mixed into the soil or is reintroduced by planting seed infected with the organism. Seed treatments or the spraying of plants is usually not very effective. Pea seed grown in semiarid regions is likely to be free from downy mildew. A 2-year rotation of crops has been suggested as one method of holding this disease in check in some localities. (See *78*, *145*.)

[8] RAMSEY, G. B. OOSPORE STAGE OF PERONOSPORA VICIAE ON PEAS. U. S. Bur. Plant Indus., Plant Dis. Rptr. 15 : 52–53. 1931. [Mimeographed.]

GRAY MOLD ROT

(*Botrytis* spp.)

Although gray mold rot may occur in peas and beans from any locality where the weather is foggy or wet and moderately cool during the shipping season, the disease is seldom of great importance on beans on the market. Peas shipped from California, Washington, and Idaho sometimes show this decay ranging from 1 to 10 percent.

The first symptoms of gray mold rot on peas are water-soaked grayish-green spots ¼ to ½ inch in diameter. No mycelium is evident on the smaller areas; but on the larger ones there is usually a moderately fine white mold, and the central area has the grayish-brown or smoke-colored mycelium and granular spore clusters that characterize *Botrytis* species.

Infection may take place throughout a temperature range from 32° to 95° F. At 32° it takes about 2 weeks for lesions to become visible, whereas at 72° they may be apparent within 2 days. At temperatures between 53° and 63° infections may take place and visible lesions develop within 4 days.

Pea pods that show small water-soaked spots should not be packed for long-distance shipment.

(See *117.*)

MECHANICAL INJURY

During the harvesting, grading, and packing of fresh green peas it is impossible to avoid some bruising and other mechanical injury. A large number of such injuries are so slight as to pass unobserved, but when the injury is severe enough to cause death of groups of cells and air is admitted into the tissues, the affected areas become conspicuous as white spots and blotches (pl. 11, *C*). Unless the epidermis is badly ruptured over the injured areas there is seldom much infection and decay resulting from such blemishes. Bacterial soft rot (p. 46) sometimes follows in bruised areas, if the peas become warm in transit.

MOSAIC

Mosaic is an important field disease of peas, and a serious market factor because of the distorted and poorly filled pods produced on affected vines. As with most virus troubles, the symptoms usually manifest are crinkling, mottling, and distortion of the leaves. Considerable variation exists because of the fact that there are several kinds of virus that affect peas. In some cases a leaf vein-clearing is characteristic; in others the leaves are spotted, mottled, and severely twisted and curled, and their food-making capacity is so reduced that the plants are stunted and produce few or no marketable pods. When the vines become severely infected before the pods are formed the pods produced often are bodly distorted and mottled by dark-green ridges and sunken yellowish-green areas (pl. 11, *E*). If any seeds are developed they are likely to be smaller and more yellow than normal.

The tall varieties of peas grown in California and Washington for the market seem to be more susceptible than the varieties grown chiefly for canning. In experimental tests the American Wonder, Perfection, Canners Gem, Dwarf White Sugar, Little Marvel, Wisconsin Early Sweet, Surprise, and several others have proved resistant to common mosaic.

The pea aphid, *Macrosiphum (Illinoia) pisi* (Kalt.), carries the virus from plant to plant in the field. The isolation of pea fields from related leguminous plants, such as clover, that may harbor the disease, and the development of resistant varieties should prove effective control measures.

(See *132*, *143*, *146*, *157*, *158*.)

POD SPOT (BLIGHT)

(*Mycosphaerella pinodes* (Berk. and Blox.) R. E. Stone)

Pod spot, also known as ascochyta blight, is one of the most common and most serious diseases of the pea and is found to some extent wherever peas are grown. All varieties of field and garden peas are affected and some closely related plants, such as vetch, are susceptible. All parts of the plant may be attacked. The disease is not only a factor from the production point of view but is also a direct factor from the point of view of marketing, because the lesions disfigure the pods and open the way for infection by secondary organisms.

This disease is characterized by long and sometimes girdling lesions on the stem. Grayish-tan circular spots are formed on the leaves. On the pods the lesions are rather sharply depressed and circular, varying from one-eighth to one-fourth inch in diameter (pl. 10, *A*). These spots are pinkish to tan in color, and in older stages may show small, black fruiting bodies (pycnidia and sometimes perithecia) in their centers. Deep lesions frequently penetrate the pod wall and discolor the seeds within. The old spots probably do not enlarge in transit although incipient lesions may develop, and spots invaded by secondary organisms sometimes enlarge during transit.

Besides the pycnidial stage of *Mycosphaerella pinodes* (*Ascochyta pisi* Lib.) there is a related fungus, *Ascochyta pinodella* Jones, which causes pod spots and blight and which may be confused with the disease caused by the first-named organism. These fungi are carried through the winter as dormant mycelium within infected seeds of the previous year or by fruiting bodies (perithecia) in dead vines. Young plants become infected and in turn produce abundant masses (pycnidia) of summer spores. These spores are disseminated and cause new infections, if warm wet weather prevails. Later in the season the perithecial fruiting bodies are developed in stem and pod lesions.

As the causal organism is carried through the winter on diseased plants, all vines insofar as possible, should be burned. Seeds also serve to carry the fungus through the winter. Western grown seed is to be recommended because it is usually free from infection. Crop rotation should be practiced in some regions.

(See *101*, *111*, *113*, *121*, *148*, *149*, *150*.)

A, Pea pod spot. *B*, Pea bacterial blight. *C*, *D*, Pea downy mildew. *E*, Pea scab.

POWDERY MILDEW

(*Erysiphe polygoni* DC.)

Powdery mildew is often seen on the market in a mild form, but the seriously affected pods of peas and beans generally are discarded by the graders and packers at the shipping point. There is an indirect effect, however, in that pods not directly infected but from diseased plants may be stunted and of poor quality. Occasionally peas and beans get on the market, even though they are actually severely blemished. Such pods have irregular brown lacelike superficial spots and blotches or small areas covered with white powdery spores (pl. 11, *D*). In the field the disease generally does not become serious, unless an attempt is made to grow peas or beans during late summer.

The fungus itself, like other powdery mildews, furnishes a positive diagnostic character. It grows on the surface of the affected tissues and forms a dense persistent mycelium, which may cover the leaves, stems, and pods. This mycelium produces in profusion the white spores (conidia) that give the fungus its powdery appearance. Badly affected organs look as though they were dusted with white powder. The plant tissues so affected cannot carry on normal photosynthesis, and as a result the plants are stunted, develop abnormally, and may even die prematurely. Often in peas and beans no fruit is set. The causal organism is largely superficial in growth, but it sends large feeding organs into the epidermal cells which cause brownish discoloration and death of small areas under the fungus pads.

Late in the season black spherical fruiting bodies (perithecia) are produced from the mycelium. These bear the spores that serve as primary inoculum in the spring.

When attacks by powdery mildew are threatened, it is advisable to dust or spray with sulfur. Variations in susceptibility to powdery mildew are shown by commercial varieties of beans and peas. This suggests the possibility of obtaining resistant varieties by selection and breeding.

(See *80*, *89*, *101*, *129*, *148*.)

SCAB

(*Cladosporium pisicolum* Snyder)

Peas from California and Washington sometimes show rather serious blemishes caused by *Cladosporium*. Dark-brown or black, irregular, raised, scablike spots sometimes occur in great numbers over the surface of the pods (pl. 10, *E*). Usually the spots do not penetrate the pod wall, but in some instances they may do so, in which case the seeds may be discolored. Seed infections appear as small blisters or brown and black spots. In advanced stages of seed infection the lesions may become dark, sunken, and covered with greenish powdery spores. The inside wall of the pods underneath these scab lesions usually shows white felty or hairlike proliferations.

In the field irregular discolored spots may be observed on the leaves, and under humid conditions a velvety growth of mold with smoke-colored spores is evident. The spots may eventually turn

brown and cause a frayed and tattered appearance of the foliage. Narrow brown to black streaks are produced on the stem, petioles, and other parts of the vine. The fruit pedicel lesions often extend over into the calyces and pod tissues. Most of the pod infections appear to begin under or near the calyces.

The organism causing this disease has a minimum temperature for development of about 37° F. The optimum temperature is around 70° and the maximum between 88° and 93°. The fungus is apparently carried from year to year in plant rubbish and in the soil and may also be carried in the seed. All varieties tested so far have proved susceptible but show varying degrees of infection.

No satisfactory control measures are yet known.

(See *144.*)

SEED SPOT

A spotting of green pea seeds, apparently due to a nonparasitic cause, has been found on the markets in shipments from several important pea-growing sections. This disease is most common in the Surprise variety and closely related types, and in crosses between Surprise and other varieties. On the market there is no way of detecting this trouble except by opening the pods and examining the seeds. The spots appear as small circular, water-soaked areas on the seed coat next to the pod wall (pl. 11, *A*). Sometimes these areas are greenish yellow and resemble the lesions caused by the bacterial blight organism. So far, however, no bacteria or fungi have been found associated with this trouble and usually not all seeds in a pod show spotting. The seeds usually are normal in size and appearance except for the slight discoloration of the seed coat next to the pod wall. Affected seeds germinate well, and may or may not produce spotted seeds in the following crop, development of the blemishes apparently depending on environmental factors or soil conditions that are not yet understood.

(See *159.*)

SPOTTED WILT

The spotted wilt virus is becoming more and more prevalent on a wide variety of vegetable crops in the field and on the market. Many of the symptoms manifested are very similar to those of mosaic and related virus troubles. See Mosaic, page 37.

Spotted wilt of peas is characterized by brown streaks on the stems and leaf veins and by brown spots on the leaves and pods. Irregular brown patterns with concentric markings may also appear on the pods, or the pods may be stunted, distorted, and collapsed. Sometimes the seeds also show brown spots.

The infective principle (virus) that causes this disease is carried from plant to plant by thrips.

(See *120*, *153.*)

THRIPS INJURY

(*Thrips tabaci* Lind.)

The feeding punctures made by thrips on pea pods sometimes cause blemishes that tend to reduce the market value of severely

A, Pea seed spotting. *B*, Pea thrips injury. *C*, Pea mechanical injury. *D*, Pea powdery mildew. *E*, Pea mosaic.

affected stock. When the insects extract the sap out of the surface cells of the pod these cells die and become grayish or silvery white. The silvery marks, streaks, and etched areas constitute a superficial blemish that is generally overlooked by the trade unless the appearance of the lot as a whole is greatly injured (pl. 11, *B*).

No decay has been observed to follow thrips injury on peas.

PARSLEY FAMILY

Within the botanical family of plants known as the Umbelliferae, or parsley family, are found in addition to many wild species a number of important cultivated plants. Chief among these are anise (*Pimpinella anisum* L.), caraway (*Carum carvi* L.), carrot (*Daucus carota* L.), celery (*Apium graveolens* L.), dill (*Anethum graveolens* L.), finocchio (*Foeniculum vulgare* L.), parsley (*Petroselinum hortense* Hoffm.), and parsnip (*Pastinaca sativa* L.). As a group they are characterized in part by the possession of abundant volatile oils. Consequently, many are used for the flavoring value of their seeds or for the flavoring and garnishing value of their leaves. Others are eaten for the flavor and food value of their leafstalks or taproots.

CARROT

The carrot (*Daucus carota* L.) is grown for its elongated edible crown tuber or fleshy taproot. The most desirable types have a slightly tapering or nearly cylindrical root of moderate size. Anything that impairs the appearance, flavor, or crispness of the root reduces the marketability of the crop.

Carrots pulled while immature are usually tied together in bunches of four or five and marketed immediately with the tops on; but sometimes they are topped. Those allowed to mature in the field are pulled, topped, and marketed either immediately or after several months in storage.

The carrot is subject to a number of diseases that affect the foliage in the field; chief among these are bacterial blight (*Phytomonas carotae* Kendrick), cercospora leaf blight (*Cercospora apii* Fres. var. *carotae* Pass.), downy mildew (*Plasmopara nivea* (Ung.) Schroet.), Macrosporium leaf blight, and yellows. (See Celery, Mosaics and other Virus Diseases, p. 53.)

Diseases that affect the root in the field include primarily bacterial soft rot, rhizoctonia crown rot, root knot (*Heterodera marioni* (Cornu) Goodey), sclerotium rot (*Sclerotium rolfsii* Sacc.). (See Beans, Sclerotium Rot, p. 30), and watery soft rot.

The most important diseases of carrots in transit, storage, and on the market are bacterial soft rot, black rot, fusarium rot, gray mold rot, rhizoctonia crown rot, rhizopus soft rot, and watery soft rot. (See *181*, *215*.)

BACTERIAL SOFT ROT

(*Erwinia carotovora* (Jones) Holland)

Bacterial soft rot of carrots is of most importance on roots that have been topped and held in farm storage, although it may occur

on the roots and tops of bunched carrots. The decay is recognized by the soft, mushy, and frequently slimy condition of affected tissues. Stored carrots may be affected anywhere on the root but frequently are attacked at the crown, with the decay extending deep into the central part of the taproot. (See Celery, Bacterial Soft Rot, p. 46.)

BLACK ROT

(*Alternaria radicina* F. Meier, Drechs., and E. D. Eddy)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Black rot has been reported on the market in New York, Massachusetts, Pennsylvania, and the District of Columbia. It is essentially a market disease of topped carrots that have been held in storage. Although reported on celery, parsley, and parsnips by certain European investigators it has been found in this country only on carrots.

The decay is readily identified by the presence of black, slightly depressed lesions at the crown or at any point over the surface of the root (pl. 12, *B*). When it occurs at the crown the decay either may be shallow or may extend deep into the central part of the root. Lesions on the side of the root are more conspicuous than those at the crown but are actually less damaging because the decay is confined to the outer tissues. Such lesions are irregular in shape, variable in size, and may occur either singly or otherwise. In all cases the affected tissues are somewhat soft and are greenish black to black in color. The characteristic black growth of the causal fungus may or may not be present on the surface of the lesions.

By artificial inoculation the disease can be produced on the foliage of the carrot plant, where it resembles in great measure the macrosporium leaf blight. (See p. 44.) In this country it has not been found occurring naturally as a leaf disease.

Black rot is responsible for considerable loss in storage and on the market, particularly in the Northeastern States. Because of its slower rate of development it is not as important as bacterial soft rot, gray mold rot, or watery soft rot. As a crown rot it renders the carrot unfit for food. Where it is found chiefly as a side rot it disfigures the root and furnishes favorable points of entry for the organisms responsible for more destructive decays.

CAUSAL FACTORS

Black rot is caused by the fungus *Alternaria radicina*. This organism is undoubtedly capable of continued existence on dead plant material in the soil. The carrot root is thus readily infected in the field. It is entirely possible that infection may likewise occur in storage cellars where diseased roots of the previous season have been stored, or where contaminated soil or plant debris has been brought in with the current season's roots.

The pathogen can penetrate the apparently unbroken skin of the root. Infection is facilitated, however, by the presence of wounds. Fresh or old wounds, either at the crown or side, together with the broken remains of small rootlets are the chief infection courts. In Europe the pathogen has been reported to be carried on the seed,

from which it spreads to the seedling, foliage, crown, and root of the plant. Nothing comparable to this has been observed in this country.

Carrot roots are susceptible to infection by the black rot fungus anywhere in an approximate temperature range from 31° to 93° F., but development is slow near the lower limit of this range. The optimum temperature for infection and decay is approximately 82°.

CONTROL MEASURES

Losses from black rot can probably be reduced by avoiding excessive bruising, particularly at the crown, during harvesting operations.

Although carrots cannot be stored below the temperature range of infection without danger of freezing, infection and decay can be retarded by storage at temperatures of 32° to 35° F.

(See *187*, *191*.)

FUSARIUM ROT

(*Fusarium* spp.)

Topped carrots held for some time at farm storage temperatures frequently become affected with a shallow spongy decay caused by *Fusarium* spp. The decay follows wounds at the crown and sides of the taproot and may be recognized by the sparse development of white mold both in cavities within the diseased tissue and over the surface of the lesion. Bacterial soft rot is frequently associated with it. Limited tests indicate that fusarium rot is of little commercial importance at temperatures below 46° F.

This type of decay is seldom found affecting bunched carrots on the market. However, there is a type of fusarium injury that occasionally constitutes an important market blemish of California bunched carrots.[9] With this injury the taproots are apparently invaded at the point of emergence of secondary roots by weakly pathogenic species of *Fusarium* and an irritation is set up which results in the formation of shallow corky or scabby areas (pl. 13, *B*). During growth and expansion of the taproot the lesions enlarge by extending around the circumference of the taproot. The lesions are of chief importance as a blemish of the taproots, although occasionally a shallow decay develops that seldom penetrates deeper than one-eighth of an inch.

(See *188*.)

GRAY MOLD ROT

(*Botrytis* spp.)

Gray mold rot is a common decay of topped carrots, parsnips (pl. 12, *A*), and to a much less extent celery when these have been held for some time in cold storage. Only occasionally is it of importance on bunched carrots or on celery that has not been stored. Infected tissues are light brown and water-soaked at first; later they may become somewhat spongy. In advanced stages of decay a grayish-white mold develops over the surface of the lesions. When conditions become favorable for spore production the decay may be readily identified by

[9] RAMSEY, G. B. FRUIT AND VEGETABLE DISEASES ON THE CHICAGO MARKET IN 1936. U. S. Bur. Plant Indus., Plant Dis. Rptr. Sup.: 101, 81–96. 1937. [Mimeographed.]

the velvety grayish-brown appearance of the abundant fruiting stalks and spore masses. Infection normally occurs through wounds.

Temperature studies have shown that the gray mold pathogen can infect carrots throughout a range extending from 32° F. to approximately 76°. Decay was greatest at 72.5°. The percentage of infection and the rate of decay were considerably reduced by storage at temperatures of 32° to 35.6°. (See also Peas, Gray Mold Rot, p. 37).

MACROSPORIUM LEAF BLIGHT

(*Macrosporium carotae* Ell. and A. B. Langlois)

Macrosporium leaf blight is rather generally distributed throughout the commercial carrot-growing sections of the country. Frequently it is of considerable economic importance in the field. On the market it is occasionally found as a leaf blemish of bunched carrots. The disease is confined to the leaves and is more severe on the older ones. The symptoms are a yellowing and browning of the tissues in areas of irregular size and shape at the margins of the leaf. Entire leaves may turn yellow, brown, and finally black, without the formation of individual scattered spots. Where the disease is widespread in the field the blighted foliage is very conspicuous. During seasons when blight is of importance, increased yields may be obtained by spraying with bordeaux mixture.

(See *163*, *169*.)

RHIZOCTONIA CROWN ROT

(*Rhizoctonia solani* Kühn; *Corticium vagum* Berk. and Curt.)

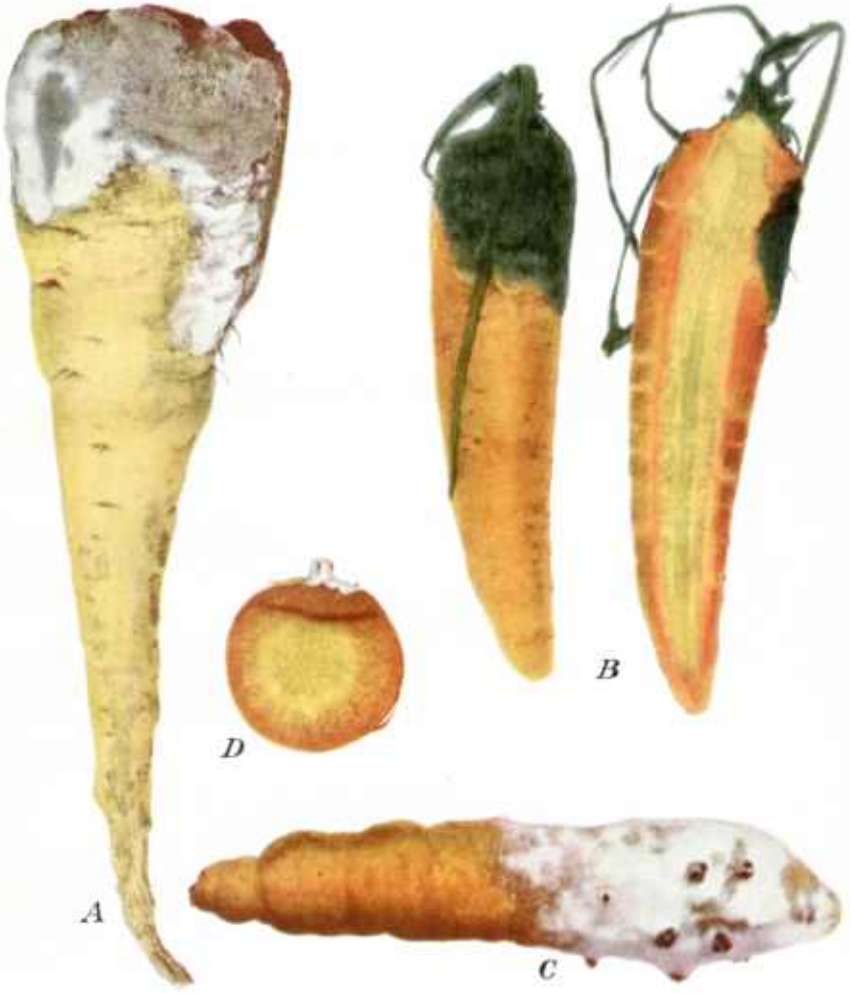
Rhizoctonia crown rot is occasionally found on carrots both in the field and in storage. As indicated by the name, the decay occurs at the crown of the plant. There the leaf petioles (particularly of the central leaves) are rotted off at the base and the leaves are killed. The decay may then advance into the root. The affected tissues of the root are brown and soft. The brown mycelium and chocolate-brown irregular sclerotia of the fungus may be present on the surface of decaying roots.

An unusual set of symptoms was once observed on New York carrots[10] held in cold storage throughout the winter. In addition to the usual symptoms of decay at the crown there were numerous small slightly depressed spots over the surface of the roots. These resembled, in a general way, the lesions of fusarium rot (p. 43). Infection had apparently taken place first in the secondary roots, from which the decay had advanced to the taproot and had continued to develop during storage.

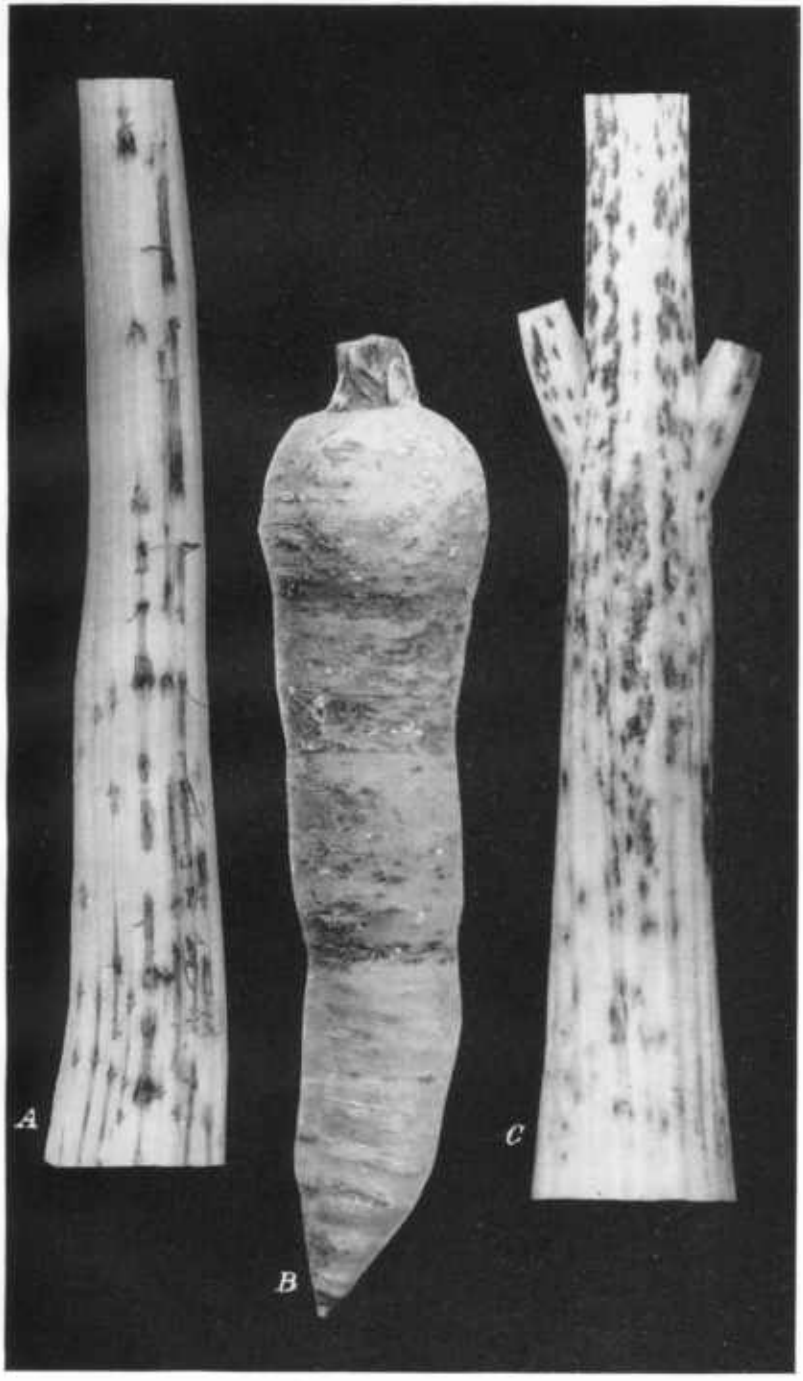
This disease is ordinarily not sufficiently important to require special control measures. Carrots from fields in which the disease was serious may be expected to show decay later on in storage, so they should be marketed as soon as practicable.

(See *163*, *221*.)

[10] RAMSEY, G. B. MARKET PATHOLOGY NOTES FROM CHICAGO. U. S. Bur. Plant Indus., Plant Dis. Rptr. 18: 40–41. 1934. [Mimeographed.]



A, Parsnip gray mold rot. *B*, Carrot black rot. *C*, *D*, Carrot watery soft rot.



A, Celery cracked stem. *B*, Carrot fusarium rot. *C*, Celery late blight.

RHIZOPUS SOFT ROT

(*Rhizopus tritici* Saito and *R. nigricans* Ehr.)

Rhizopus soft rot is a common transit and market decay of either bunched or topped carrots and is frequently of importance on topped carrots in common or farm storage. The affected tissues have a brownish water-soaked appearance and are soft and watery. The early stages of decay are distinguished from those of bacterial soft rot by the presence of the fungus threads, which can readily be demonstrated by gently pulling apart the diseased tissues. In later stages of decay the coarse white mycelium of the pathogen can be found growing over the surface of the lesions. The black fruiting bodies of the causal fungus may or may not be present.

Extensive storage tests have shown that *Rhizopus nigricans* is of much less importance on carrots than is *R. tritici*. The optimum temperature for the development of decay of carrots by the latter species was found to be around 90° F.; no decay developed in roots stored at temperatures lower than 66°. In fact, seldom did any type of rhizopus soft rot, resulting from natural infections, occur below 54°. The decay is of most importance on carrots that have been subjected to unusually high temperatures. (See Beans, Rhizopus Soft Rot, p. 27.)

(See *188*.)

WATERY SOFT ROT

This disease is illustrated in plate 12, *C*, *D*. (See also Celery, Watery Soft Rot, p. 56.)

CELERY

Celery (*Apium graveolens* L.) is grown for its leafstalks. The celery plant consists of a short thickened stem or crown on which are borne the fibrous root system below and the succulent leafstalks above. The latter grow in whorls about a terminal bud within the central part or "heart" of the plant. Each leafstalk bears three pairs of leaflets and a terminal leaflet. Celeriac (*Apium graveolens* L. var. *rapaceum* DC.), also known as turnip-rooted celery, is a form in which the leaves are borne on a thickened, turniplike crown that is the edible part of the plant.

The marketability of celery depends chiefly upon its tenderness, crispness, and color, and its freedom from blemishes and decay. Tenderness is determined to a great extent by the variety, the local growing conditions, and the weather during the growing season. Lack of crispness may be due to excessive transpiration or drying out during the period of growth, harvest, transportation, or storage. Color is determined by the variety, the method of blanching used, and the duration of the blanching process.

Most of the injuries and diseases to which the celery plant is subject not only occur in the field but are of direct importance on the market either as blemishes or as decays of the leaves or the leafstalks. These include bacterial soft rot, bacterial blight, black-heart, brown stem, cracked stem, early blight, freezing injury, late blight, phoma root rot, tarnished plant bug injury, and watery soft rot.

Fusarium yellows (*Fusarium apii* Nelson and Sherbakoff and *F. apii* var. *pallidum* Nelson and Sherbakoff (*193*), rootknot, and mosaic are primarily field diseases; with the occasional exception of the latter they are market factors only indirectly through stunting of the plants.

Gray mold rot is primarily a transit and storage trouble.

BACTERIAL BLIGHT

(*Phytomonas apii* (Jagger) Bergey et al.)

Bacterial blight of celery has been reported from Indiana, Michigan, Minnesota, New York, Ohio, and Pennsylvania, and probably occurs elsewhere in the northern celery-growing regions of the country. The development of the disease is favored by periods of warm rainy weather. Celery harvested from diseased fields is occasionally found on the market with the leaves blemished by the characteristic bacterial blight lesions.

The chief symptom of the disease is the development of numerous small, irregularly circular spots on the leaflets. At first these are yellow, later turning to a rusty brown with a yellow border or halo around them. They may remain as numerous individual spots, or they may coalesce to cover large areas of the leaflets. The spots differ from those of early blight (p. 50) and late blight (p. 51) in that neither ashen-gray mold nor black fruiting bodies are present.

The disease can be successfully controlled in the seedbed and in the field by spraying with bordeaux mixture or dusting with copper-lime dust. (See Celery, Late Blight, p. 51.)

(See *170*, *179*, *194*, *223*.)

BACTERIAL SOFT ROT

(*Erwinia carotovora* (Jones) Holland)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Bacterial soft rot is an important market disease of a wide variety of vegetable commodities. Wounded or bruised tissues are readily affected and the causal organism is a secondary invader of tissues weakened or killed by other diseases. On celery the decay commonly follows such troubles as late blight, early blight, freezing injury, black-heart, and bruises and injuries of all sorts; it frequently is of considerable importance on stock that has not been promptly stored or shipped after harvest.

The disease at times causes heavy field losses. If only the outer leaves are affected these can be stripped off during harvesting and packing with only a minimum of loss. Occasionally, however, the disease occurs as a crown rot or heart rot, in which case the plant soon becomes commercially worthless and serious losses are incurred.

The first symptoms of the decay are a water-soaked appearance and softening of the affected tissues. These areas soon turn brown and become very mushy but the epidermus remains intact (pl. 14, *A*, *B*). Although in some vegetables the decayed tissues have a slimy consistency and an offensive odor, in celery neither symptom is ordi-

A, Celery early blight. *B*, *C*, Celery late blight.

narily characteristic. The decay may affect the crown, the leafstalks, or the leaflets. The decayed areas may be continuous or scattered over the plant. The rot develops rapidly under warm, humid conditions and serious market losses often occur in infected stock.

CAUSAL FACTORS

Bacterial soft rot of celery and other vegetables is caused by *Erwinia carotovora* and other closely related species of bacteria. The causal organisms are all soil inhabitants and will grow on a wide variety of plant products. They are readily carried over from season to season on stored stock and are found as contaminants of plant debris in places where diseased stock has previously been stored. The practice of allowing plant trash to accumulate aids in increasing the number of bacteria that are readily available for dissemination to growing or harvested stock. These are undoubtedly spread by tools, insects, domestic animals, and running or splashing water. Inoculation may result from direct contact with infested soil or infested plant trash. Plants that become infected early in the season serve as a ready source from which bacteria are spread later in the field.

Celery plants may become inoculated during harvesting and packing operations through contact with contaminated cutting knives, hands, tools, and wash water. Although the decay may be spread in transit and storage through contact with diseased plants, much of what appears there undoubtedly had its inception in the field.

The causal organisms enter the plant through wounds, insect punctures, and lesions of other diseases. Moisture is essential for the growth and reproduction of the bacteria, and they are sensitive to desiccation and direct sunlight. Wet weather usually favors the decay by increasing the rate of multiplication and dissemination of the causal organisms and by moistening the plant tissues. It has been found in Minnesota, however, that celery is more susceptible to heart rot in the field during dry periods. This is believed to be due to variation in the activities of the insect carrier during periods of dry weather.

The decay is usually favored by high temperatures provided the humidity is high. Under conditions of high humidity the most favorable temperature for decay is in the range of 77° to 86° F., although there is a wide temperature range within which the decay may progress actively. At temperatures of about 45° or lower the decay is checked.

CONTROL MEASURES

Control measures include the adoption of general sanitary practices around storage houses, pits or cellars, and during harvesting and packing operations. Any practice that will reduce the likelihood of contamination by the soft rot bacteria should be of help in reducing losses from the disease.

The most important measure for the control of bacterial soft rot is that of temperature control during transit and storage. Consequently promptness in placing the celery under desirable transit or storage temperatures is advantageous, particularly if the celery would

otherwise be subjected to warm, humid conditions. Once the stock has become thoroughly cooled the decay should make little progress during transit. Precooling either before or after loading reduces the likelihood of decay.

(See *160*, *177*, *180*, *189*, *212*, *225*, *226*.)

BLACK-HEART

OCCURRENCE, SYMPTOMS, AND EFFECTS

Black-heart is found in all sections of the country where celery is grown commercially. It is probably of most importance in the Florida and California crops but has frequently been found to a serious extent in other States.[11] Although primarily a field disease it is at times of considerable importance on the market.

The first symptom of black-heart is the appearance of brown lesions on the outer margins of the youngest and tenderest leaves at the center of the celery plant. The affected tissues in these lesions may dry out, retain their brown color, and remain sharply set off from the healthy tissues; the plant may then recover and continue growth. This may occur several times during the growing season. Or there may be a darkening of the affected tissues accompanying the extension of the lesions throughout the leaflets and petioles of the heart leaves so that the entire central part of the plant is blackened and killed (pl. 15).

In the more advanced stages of black-heart the affected tissues are frequently invaded by various secondary soil organisms and a soft rot develops. Black-heart should not, however, be confused with the "heart rot" or "crown rot" types of bacterial soft rot. (See Bacterial Soft Rot, p. 46).

Although occasionally the outer leaves become yellowed or chlorotic before other symptoms of black-heart appear, usually there is no external evidence of the disease even though the early symptoms could be demonstrated by cutting open the plant. This, together with the fact that slight external evidence of the disease may be overlooked, makes possible the occurrence of advanced stages of black-heart on the market in celery that was presumed to be free of the disease at the time of shipping. Black-heart may become more serious during transit or on the market, but it originates in the field.

CAUSAL FACTORS

Although considerable information has been obtained regarding the conditions that favor the appearance of black-heart it cannot be said that the cause is fully understood. Undoubtedly the disease is the result of a very complex physiological disturbance in the plant.

Development of black-heart usually follows periods of drought or of excessive soil moisture. It has been suggested that these extremely different conditions may have the common effect of killing the root hairs. Once the soil-moisture conditions approach a point favorable for the occurrence of the disease, any factor that increases the rate of

[11] FOSTER, A. C. BLACKHEART DISEASE OF CELERY. U. S. Bur. Plant Indus., Plant Dis. Rptr. 18: 177–185. 1934. [Mimeographed.]

Celery black-heart.

growth appears to increase the likelihood of black-heart development. Thus, although the form of nitrogen used in the fertilizer has little effect, the greater the amount used the greater will be the amount of black-heart if moisture conditions are favorable for its appearance.

The disease is more severe after periods of high temperature and is of much more importance as the celery reaches maturity than earlier in the growing season.

CONTROL MEASURES

In celery grown under irrigation black-heart has been successfully controlled by careful regulation of the irrigation waters. Excessive soil moisture immediately prior to harvesting is to be avoided particularly. Only moderate amounts of nitrogen fertilizer should be used. Harvesting the crop promptly as it approaches maturity may reduce losses. Some measure of control has been secured by growing partly resistant types of celery.

Where any evidence of black-heart has occurred in the field particular care should be taken to avoid packing affected plants, for they are practically certain to develop bacterial soft rot (see p. 46) in transit.

(See *172*, *173*, *206*, *214*, *224*.)

BROWN STEM

The symptoms of brown stem are a browning and pitting of the tissues immediately beneath the epidermis of the outer leafstalks. Although the cause is unknown it appears to be nonparasitic. Where brown stem occurs it usually is found on celery that has been allowed to remain in the field for some time after having reached maturity. At such times it frequently is associated with black-heart.

Brown stem is of importance as a blemish of celery both in the field [12] and on the market.[13] The affected areas of the leafstalks probably also serve as points of entry for organisms causing decay in transit and on the market.

(See *173*.)

CRACKED STEM

Cracked stem has been of major importance in Florida and of occasional importance in a number of other States. It is found from time to time on the market, where it frequently causes serious losses.

The disease is first recognized by a brownish mottling of the leaves. The roots are also affected, turning brown and dying back from the tips. The leafstalks become brittle, and brown longitudinal streaks appear over the ribs or vascular bundles. Transverse cracks then appear in the epidermis over these streaks and the torn epidermis curls and peels back from the cracks, giving the leafstalks a roughened appearance (pl. 13, *A*). Although the cracks are shallow and narrow they usually occur in such abundance as to render the stalk unsightly, hence of little market value.

---

[12] MACMILLAN, H. G., and PLUNKETT, O. A. PLANT DISEASES OBSERVED IN SOUTHERN CALIFORNIA IN 1936. U. S. Bur. Plant Indus., Plant Dis. Rptr. 21: 76–79. 1937. [Mimeographed.]

[13] BRATLEY, CYRIL O. NEW YORK MARKET PATHOLOGY NOTES. U. S. Bur. Plant Indus., Plant Dis. Rptr. 15: 50, 1931. [Mimeographed.]

Cracked stem is undoubtedly a nutritional disorder. In the acid muck soils of Florida the disease has been controlled by soil applications of commercial borax. On heavily limed [14] or otherwise alkaline soils in several Northern States beneficial results were obtained by applications of soil-acidifying agents, such as sulfate of ammonia, manganese sulfate, or sulfur; boric acid and borax were successfully used there on both acid and alkaline soils.

(See *173*, *192*, *200*, *201*.)

EARLY BLIGHT

(*Cercospora apii* Fresen.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Early blight is of common occurrence in all celery-growing districts of the country and is frequently of great importance in California and Florida. Losses vary considerably within a given area from season to season. They may range in severity from a blemishing of the leaves to almost total destruction of the crop.[15] On the market the disease is primarily of importance because of the less attractive appearance of infected celery and because the presence of the lesions facilitates infection by soft rot bacteria.

The first symptom of early blight is the appearance on the leaves of small pale-yellow spots which enlarge and become roughly circular. Where the spots are numerous they coalesce to form large irregularly shaped lesions. The color of the spots changes from yellow to brown, and finally, by the growth of the pathogen over the affected surface, to ash gray. The lesions are most abundant on the leaflets although they are found frequently on the leaflet petioles and occasionally on the main leafstalks, where they appear as brown longitudinal streaks that later turn gray and then nearly black (pl. 16, *A*). In most cases the lesions can be distinguished from those of late blight by the absence of the small black, fruiting bodies (pycnidia) so characteristic of the latter disease.

CAUSAL FACTORS

Early blight is caused by the fungus *Cercospora apii*. The pathogen is capable of living over from crop to crop in diseased plant refuse in and on the soil. Under favorable conditions the spores are produced there in great abundance and are carried by air currents to the leaves of growing healthy plants. The most favorable conditions for spore germination are the presence of moisture on the leaf surface and a temperature of 81° to 84° F. The optimum temperature for growth of the fungus is 77° to 86°, although it grows well between 73° and 86°. During periods of high temperature with abundant moisture the disease develops rapidly throughout the field. Periods of continued warm days followed by cool nights, with the formation of heavy dews that remain long on the plant the following day, are particularly conducive to severe losses from the disease.

[14] BOYD, O. C. VEGETABLE DISEASES IN MASSACHUSETTS IN 1936. U. S. Bur. Plant Indus., Plant Dis. Rptr. 20 : 333–337. 1936. [Mimeographed.]

[15] WELLMAN, F. L. AN UNUSUAL OUTBREAK OF CELERY EARLY-BLIGHT. U. S. Bur. Plant. Indus., Plant. Dis. Rptr. 16 : 43–45. 1932. [Mimeographed.]

A, *B*, Celery bacterial soft rot. *C*, Celery watery soft rot.

There is no indication that the spots enlarge or that new ones develop during the transit period.

CONTROL MEASURES

Early blight can usually be controlled by the program of spraying and dusting discussed more fully under late blight of celery (p. 51). (See *182*, *217*, *223*.)

FREEZING INJURY

Celery that has been severely frozen can readily be recognized by the flabby water-soaked condition of the leaves and leafstalks. Frozen leaves if not attacked by soft rot bacteria dry out and become papery; leafstalks may either become decayed or turn brown and shrivel.

There has also been recorded a second type of freezing injury characterized by the appearance of isolated sunken lesions on the leafstalks. In shape these are elliptical on the convex side and circular on the concave side of the stalk. The affected tissues soon turn dark brown, so that the lesions are very conspicuous.

Inasmuch as both of the above types of injury are readily apparent at the time of harvest it is unlikely that they will be of much importance on the market. However, celery that has been only mildly or partially frozen and that has recovered without any markedly conspicuous effects of the injury is frequently seen on the market. The only symptom by which this slight injury can be recognized is the loosening of the epidermis. This can readily be detected by slightly twisting the affected leafstalks. Then as the thin epidermal layer rises in irregular corrugations it will be seen that it has separated from the underlying tissues.

(See *175*, *222*.)

GRAY MOLD ROT

(See Carrot, Gray Mold Rot, p. 43, and Peas, Gray Mold Rot, p. 37.)

LATE BLIGHT

(*Septoria apii-graveolentis* Dorogin and *Septoria apii* (Briosi and Cav.) Chester)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Late blight is one of the most important diseases of celery and is found throughout the country wherever the crop is grown. All of the above-ground parts of the plant may be affected. Unless the disease is well controlled in the field, plants may be stunted or rendered worthless for sale. Less serious losses may result from the greater amount of trimming required and from the less attractive appearance of the marketed product. The disease is of particular importance in transit and storage because the organisms causing watery soft rot and bacterial soft rot readily enter through the late blight lesions and cause serious market decays of affected plants.

The disease is found only on celery and celeriac, although a fungus closely related to those causing late blight is responsible for a leaf spot of parsley.

There are two types of late blight, the large-spot form and the small-pot form (pl. 16, *B* and *C*). Although both are widely distributed, the latter is the more common in this country. The first symptom of the small-spot type on the leaflets is the formation of small yellowish (or chlorotic) spots. As the spots enlarge the affected tissues turn brown and then nearly black. The edges of the spots are indefinite. The diameter of the spot varies somewhat, with an average of around one-sixteenth of an inch and an upper limit of about one-eighth of an inch. Where the spots are numerous and therefore close together they may coalesce to form large brownish-black areas. The spots are further characterized by the presence of numerous black granular bodies of pin-point size, which are the fruiting bodies (pycnidia) of the pathogen. These are present even in early stages and are found either singly or in clusters over the entire surface of the spots; they frequently occur in the apparently healthy green tissues bordering the spots. The leaflet petioles and the main leafstalks are also attacked. There the lesions are longitudinally elongated and the affected tissues are brown, with abundant fruiting bodies (pycnidia) scattered over their surface (pl. 13, *C*). The lesions on the leafstalks are most numerous on the outer surface of the older ones.

In the large-spot type of late blight the spots are larger, with an average diameter of around one-half inch, more regular in outline, and have more definite boundaries. They are brown in the center and have a darker reddish-brown border. The pycnidia are not abundant, frequently develop late, and are confined to the center of the spot. Where pycnidia have not yet appeared the spots closely resemble those of early blight (see p. 50). The occurrence on the leafstalks of this type of late blight has not been reported. Possibly it occurs there with symptoms indistinguishable from those of the small-spot type of the disease.

CAUSAL FACTORS

The small-spot type of late blight is caused by the fungus *Septoria apii-graveolentis* and the large-spot type by *Septoria apii*. Both fungi are capable of living over from season to season in the seed, and in diseased celery refuse in the soil. Celery plants are thus subject to attack from the first stages of growth so that unless proper control measures are taken a high percentage of the seedlings may become diseased in the seedbed. In the field the disease may originate from spores produced either on the diseased seedlings or on plant refuse in the soil. The dissemination of spores by drainage and irrigation waters, by wind, by farm animals and machinery, and by man may cause the further spread of the disease throughout the field.

Late blight is generally considered a cool-weather disease. This is borne out by the fact that the organism responsible for the small-spot type of the disease, which is the more prevalent of the two types, grows best at a temperature range of approximately 64° to 72° F. Above 75° growth of the pathogen is very slow. The fungus responsible for the less prevalent large-spot type of the disease has an optimum temperature of approximately 72° to 75° and makes

moderate growth even up to 80°. Information on either the growth of the pathogen or the development of the disease at low temperatures is meager. Observations suggest that celery apparently free from the disease at the time of shipment may show it later on. In one experiment, however, badly diseased celery stored at 31° for 10 weeks showed no appreciable increase in the amount of late blight during the storage period.

CONTROL MEASURES

Several practices are involved in the successful control of late blight. Field sanitation is one of them. Trimmings from diseased plants should not be left in the field nor allowed to accumulate where they might later reach the field. The seedbed should receive particular attention in this respect.

The causal fungi of late blight can remain viable in infected seed for 2 years. Seed that is not older than this may be treated with hot water, calomel, formaldehyde, or corrosive sublimate to eliminate this source of infection. The value of seed treatment may, however, be questioned in view of the wide infestation of celery soils.

Spraying with bordeaux mixture or dusting with copper-lime dust is undoubtedly the most important control measure. Here again, special attention should be given to the seedbed, with as early and as frequent applications of the fungicide as appear necessary. The spraying or dusting program should be continued in the field throughout the season.

Transportation, storage, and market losses can be reduced by the control of the disease in the field. However, if infected celery is marketed it is important to keep it at as low a temperature as is practicable and to move it through the channels of trade as rapidly as possible.

(See *161*, *164*, *183*, *194*, *204*, *205*, *207*, *223*.)

MOSAICS AND OTHER VIRUS DISEASES

There are several mosaic diseases of celery. Although differing in other respects all produce a mottling of the affected leaves. These mosaics are caused by various plant viruses, which for the most part are spread from plant to plant by sucking insects.

One form of celery mosaic found in the Northeastern and North Central States is characterized by a stiffness and erectness of the affected plants. The leaves are distorted by uneven puckering and become filiform in shape. The disease appears to be caused by the common cucumber mosaic virus. Ordinarily the losses from this type of mosaic are not great, and the disease is of no importance on the market.

Another type is that known as southern celery mosaic. Plants affected with this disease appear stunted and the leaves are conspicuously yellowed and mottled. As growth continues, however, the new shoots are more erect and the mottling is often faint and not readily noted after blanching. The leafstalks often show brownish or buff-colored, sunken, translucent spots and such stalks eventually

may become brown and shriveled. The disease is of considerable economic importance in Florida and it is occasionally found affecting Florida celery on the market. It also affects carrot, dill, and parsley, as well as a wide variety of other plants in Florida. What is apparently the same disease has been reported on celery in several other States.

The most important mosaic disease of California celery is the western celery mosaic. It differs from the southern celery mosaic in that the causal virus can be transmitted only to members of the parsley family. Although the two diseases have many symptoms in common they differ in some respects. Thus the rust-colored necrotic spots and streaks that are characteristically present on the upper surface of older leaves on plants affected with the western celery mosaic have not been reported for southern celery mosaic. At the same time the brownish water-soaked spots and sunken streaks found on the leakstalks of plants affected with southern celery mosaic have not been observed on plants affected with western celery mosaic.

Calico, crinkle-leaf, and yellow spot, all of which produce chlorotic spots on the leaves of affected plants, have likewise been described from California celery; and poisonhemlock ringspot has been experimentally transmitted to celery.

There are several other virus diseases found on celery, carrots, and related plants. Thus yellows, caused by the aster yellows virus, is frequently of importance on carrots in the field. Although attempts to transmit the virus to celery have been unsuccessful it has been transmitted to anise and dill, and is responsible for a yellows disease of parsnip. In California, however, celery is affected with a yellows disease that can be transmitted to aster, carrot, parsnip, parsley, and other plants. The disease has been called California aster yellows to distinguish it from aster yellows, which as noted above cannot be transmitted to celery. Finally, there is the curly top disease in the States west of the Rocky Mountains, which affects sugar beets and a wide variety of other crop plants including carrot and which has been experimentally transmitted to celery; and there is the spotted wilt disease found affecting celery in California.

Control of the several virus diseases is based upon such measures as eradicating the weed hosts that perpetuate the disease from season to season and serve as the source of virus infection for the new crop, controlling the insect carriers, and planting resistant varieties or strains. The virus diseases are of importance on the market only when an attempt is made to market plants seriously affected with this kind of disease in the field.

(See *168*, *174*, *184*, *185*, *186*, *196*, *199*, *208*, *209*, *210*, *211*, *216*, *218*, *219*, *220*.)

PHOMA ROOT ROT

(*Phoma apiicola* Kleb.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Phoma root rot has been reported from Michigan, Ohio, and New York. Although found in plantings only on celery the disease has been produced by artificial inoculation on caraway, carrot, parsley,

and parsnip. Dill was not affected. In Europe it is known chiefly as a disease of celeriac.

Losses from phoma root rot are sporadic and largely confined to the field. In several instances, however, the disease has been responsible for approximately a 50-percent loss either in storage or on the market.

The field symptoms of the disease are wilting, yellowing, and dying of the outer leaves, followed either by stunting or by the death of the plants. The decay usually begins near the base of the leafstalks and progresses through the crown, affecting the roots in close proximity thereto. Affected roots turn brown and soon disintegrate. The diseased tissues of the crown and leaflet petioles are first bluish green and later black. The decay never extends far up the leafstalk or far down the root system, being chiefly confined to the crown of the plant. Where the disease is severe the affected tissues are usually completely disintegrated by secondary soft rot organisms.

As observed on celery in storage and on the market the decay is likewise confined to the basal part of the plant. Unless other decays are also present the affected tissues are only moderately softened and are black with a greenish-black color at the advancing edge of the lesion.

CAUSAL FACTORS

Phoma root rot is caused by the fungus *Phoma apiicola*. The pathogen overwinters in plant debris in the soil and occasionally is carried over from season to season on the seed. The disease frequently originates in the greenhouse or coldframe, but it may originate directly in the field.

The black fruiting bodies (pycnidia) of the fungus are formed in abundance over the decayed areas of the crown. The spores released from the pycnidia are carried by surface water to nearby healthy plants. The fungus is able to penetrate directly the healthy tissues, particularly in young plants.

The most favorable temperature for the growth of the pathogen is within the approximate range of 61° to 68° F. The maximum temperature for growth is around 82°. It is therefore essentially a low-temperature organism. The disease is important in the field only during the spring and fall, and is most severe during unusually cold and wet seasons. Celery that was apparently healthy when placed in commercial storage at 32° has been known to show a high percentage (18 to 90 percent) of phoma root rot when it was removed 7 to 9 weeks later. The fact that 45 percent of the plants in one carlot were affected with this decay on the market [16] likewise suggests that healthy-appearing celery from diseased fields may be already inoculated with the pathogen at the time of packing and consequently exhibit heavy losses after storage.

CONTROL MEASURES

No way is known of insuring complete control of the disease. Where it is prevalent, care should be taken to use disease-free seed

[16] WIANT, JAMES S. NEW YORK MARKET PATHOLOGY NOTES. U. S. Bur. Plant Indus., Plant Dis. Rptr. 16 : 1–2. 1932. [Mimeographed.]

and to avoid the use of seedlings from seedbeds where the disease is in evidence. Field sanitation and crop rotation are also recommended.

Celery from fields in which phoma root rot has appeared should not be stored longer than is commercially necessary, and when stored it should be examined periodically for evidences of decay. Heavy losses may occur in crated celery during storage if it shows any evidence of this decay prior to storage.

(See *162*, *176*, *195*.)

PITHINESS (HOLLOW STEM)

Pithiness is a condition of celery characterized by the central tissues of the leafstalks being soft and spongy. Frequently the affected tissues are pulled apart so that the leafstalks are hollow.

There appear to be two types of pithiness. One type is a hereditary condition and is therefore associated with certain strains and varieties. It regularly affects all leafstalks of the plant throughout the season. The second type is not hereditary but is apparently the result of unfavorable growing conditions. It may, therefore, be found in varying amounts in different seasons and localities regardless of the variety grown. This type of pithiness, identical in appearance with the first type, is usually confined to the outer leafstalks and frequently is more important late in the season as the crop approaches maturity. It is this type of pithiness that is frequently found on the market associated at times with brown stem.

(See *171*, *173*.)

TARNISHED PLANT BUG INJURY

During certain seasons considerable loss occurs in the northern celery-growing sections of the country through attacks by the tarnished plant bug (*Lygus pratensis* (L.)). The injury is produced by the insect during the feeding process, which involves the puncturing of the leafstalk and the sucking of the plant juices. Feeding usually occurs near the upper leaf joint, as the result of which the tissues in the center of the stalk at and near the joint become brownish black. Because of this the injury is commonly known as "black joint." The leaflets above the point of injury become chlorotic and wilt. Tarnished plant bug injury is occasionally found on the market on celery from the Northeastern and North Central States.

(See *166*, *167*, *206*, *213*.)

WATERY SOFT ROT

(*Sclerotinia sclerotiorum* (Lib.) DBy. and other *Sclerotinia* spp.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Watery soft rot, known also as pink rot and as sclerotinia rot, is the most important decay of celery in transit, in storage, and on the market. It also occurs on beans, peas, carrots, finocchio, parsley, parsnip, and in fact on practically all vegetables, with the notable exception of potato and onion. In the field it is frequently serious

on celery and a wide variety of other vegetable crops and is found wherever they are grown. It may occur as a damping-off of young plants or as a basal rot of the stem and leaves of older plants. Occasionally it is found on the upper parts of the plant several inches from the ground.

The symptoms of watery soft rot on market products are water soaking and softening of the affected tissues. The color changes vary with the commodity; in celery, finocchio, and other light-colored vegetables the decaying tissues are light brown with a pinkish-brown border (pl. 14, *C*). There is no characteristic odor. The decay occurs frequently at the base of the leafstalks of celery and finocchio, although it occurs as well elsewhere on the leafstalk or on the leaflet petioles. On bunched carrots it is of more frequent occurrence on the taproots than on the tops. The most important diagnostic character in the advanced stages of decay on all commodities is the presence of the white cottony mold growth of the pathogen. In hampers of peas and beans this is responsible for the so-called nests of decay, in which decayed individuals are covered and held together by the mold. In later stages of decay the resting bodies or sclerotia of the causal fungus are formed either over the surface or in cavities within the affected commodity. These are white at first, then bluish, and finally black. They are particularly abundant under field or storage conditions. There is usually considerable leakage from decaying tissues along with a shriveling of affected plant parts so that the final stage of decay, particularly in root crops such as carrots and parsnips, may be the formation of a wrinkled dry mummy.

CAUSAL FACTORS

Watery soft rot is caused chiefly by the fungus *Sclerotinia sclerotiorum*. At times, however, either *S. minor* I. C. Jagger or *S. intermedia* Ramsey may be responsible for the decay. These fungi live over from crop to crop as sclerotia either on diseased plant refuse or in the soil. Under favorable moisture and temperature conditions mycelium develops from them and spreads out through the soil to attack young plants of the current crop. Under conditions favorable for their development, small mushroomlike structures, known as apothecia, may be formed from sclerotia buried in the soil. These fruiting bodies of the pathogen push up through the soil and produce innumerable spores that are discharged and are carried by air currents to healthy plants.

The presence of moisture is essential for the germination of the sclerotia, the growth of the mycelium, the production of the apothecia, and the infection of plants. Spore production and discharge are favored by moderately low temperatures and are prevented at temperatures above 80° F. Where moisture and temperature conditions are ideal for spore development the disease may become widespread throughout the field. Such conditions apparently occurred in 1936 in the Sarasota region of Florida.[17] Following a period of cold wet weather favorable for the development of the

[17] TISDALE, W. B., and KELBERT, DAVID, G. A. PINK ROT OF CELERY IN FLORIDA. U. S. Bur. Plant Indus., Plant Dis. Rptr. 20: 134–135. 1936. [Mimeographed.]

pathogen but unfavorable for the growth of celery, a loss of 50 to 80 percent of the crop was sustained. A contributing factor was the occurrence of frost damage on the outer leaves.

Decay may be expected to develop in transit or storage in celery or other vegetable crops harvested from fields in which the sclerotinia disease was an important factor, even though they appeared to be free of decay at the time of harvest. Particularly is this true if the commodity was wet when it was put into transit or storage.

Wounds are not essential for the entrance of the pathogen, but the presence of fresh wounds favors immediate infection and may result in an increased amount of decay. Plants not inoculated with the pathogen previous to the time of harvest will not become infected unless they later come into contact with infested soil or diseased plants.

Each of the species of *Sclerotinia* has a wide temperature range for growth and for production of decay. Infection may occur at temperatures as low as 32° to 34° F. and as high as 82°, with an optimum of 70° to 78°. Low temperatures will retard the development and spread of the decay in transit and storage but will not completely control it. During long storage periods severe losses are frequently noted at temperatures between 32° and 45°.

CONTROL MEASURES

There is no way of completely controlling watery soft rot. Field sanitation practices which eliminate diseased plants and plant trash from the field will tend to reduce the number of sclerotia and so reduce the likelihood of infection.

Celery harvested from fields where the disease is serious should be trimmed carefully, shipped under as low a temperature as is practicable, and sold for consumption as soon as possible.

All vegetable commodities susceptible to watery soft rot should be put into transit or storage in a dry condition and held at temperatures as low as is practicable.

(See *178*, *188*, *197*, *198*, *202*, *203*, *204*.)

FINOCCHIO

Finocchio (*Foeniculum vulgare* Hill. var. *dulce* Alef.), frequently but erroneously [18] called "anise" or "sweet anise" on the market, is grown and marketed for the edible bulblike enlargement found at the base of the above-ground portion of the plant. This is formed by the compact and overlapping arrangement of the thickened leafstalk bases and appears as a well-branched fairly solid structure.

Few diseases have been reported on finocchio. On the market it has been found affected only with bacterial soft rot and watery soft rot.

---

[18] The term "anise" has long been recognized in horticultural literature as the common name of an entirely different plant, *Pimpinella anisum* L. It has likewise been applied at times to sweet cicely (*Myrrhis odorata* Scop.), another member of the family Umbelliferae. Its usage as a synonym for finocchio is unfortunate and should be discouraged.

BACTERIAL SOFT ROT

(See Celery, Bacterial Soft Rot, p. 46.)

WATERY SOFT ROT

(See Celery, Watery Soft Rot, p. 56.)

Parsley

Parsley of the market consists of the leaves of the parsley plant (*Petroselinum hortense* Hoffm.). These are marketed in bunches and used for flavoring and garnishing foods. Freshness, crispness, good green color, and freedom from blemishes and decay are desired market qualities. A variety with crisp curly leaves, known as curly parsley, is similarly marketed and used. Another variety, known either as rooted parsley or as turnip-rooted parsley, is marketed as a thin long taproot with leaves attached.

Diseases of parsley in the field include bacterial soft rot, downy mildew (*Plasmopara nivea* (Ung.) Schroet.), rootknot, septoria blight (*Septoria petroselini* Desm.), and watery soft rot.

Bacterial soft rot and watery soft rot are the important transit and market diseases of parsley.

(See *190.*)

BACTERIAL SOFT ROT

(See Celery, Bacterial Soft Rot, p. 46.)

WATERY SOFT ROT

(See Celery, Watery Soft Rot, p. 56.)

Parsnip

The parsnip (*Pastinaca sativa* L.), like the carrot, is grown for its edible, fleshy taproot. The plants are allowed to mature in the field, and afterwards they are pulled, topped, and the roots usually stored before they are marketed.

Diseases that affect the foliage in the field include among others cercosporella leaf spot (*Cercosporella pastinacae* Karst.), downy mildew (*Plasmopara nivea* (Ung.) Schroet.), powdery mildew (*Erysiphe polygoni* DC.), and ramularia leaf spot (*Ramularia pastinacae* Bubák). Field diseases of the roots include root knot, scab (*Actinomyces scabies* (Thax.) Güssow), and watery soft rot.

The most important diseases of the parsnip during transit, storage, and marketing are bacterial soft rot, gray mold rot, and watery soft rot.

(See *165.*)

BACTERIAL SOFT ROT

(See Celery, Bacterial Soft Rot, p. 46.)

GRAY MOLD ROT

This disease is illustrated in plate 12, *A*.

See also Carrot, Gray Mold Rot, page 43, and Pea, Gray Mold Rot, page 37.

WATERY SOFT ROT

(See Celery, Watery Soft Rot, p. 56.)

LITERATURE CITED

LILY FAMILY

(1) ANDERSON, P. J.
1921. DEVELOPMENT AND PATHOGENESIS OF THE ONION SMUT FUNGUS. Mass. Agr. Expt. Sta. Tech. Bul. 4, pp. [99]–133, illus.
(2) ———
1924. SUSCEPTIBILITY OF SPECIES OF ALLIUM TO ONION SMUT. (Abstract) Phytopathology 14: 26.
(3) ANGELL, H. R.
1929. PURPLE BLOTCH OF ONION (MACROSPORIUM PORRI ELL.). Jour. Agr. Res. 38: 467–487, illus.
(4) ——— WALKER, J. C., and LINK, KARL PAUL.
1930. THE RELATION OF PROTOCATECHUIC ACID TO DISEASE RESISTANCE IN THE ONION. Phytopathology 20: 431–438.
(5) ARK, P. A., and BARRETT, J. T.
1938. PHYTOPHTHORA ROT OF ASPARAGUS IN CALIFORNIA. Phytopathology 28: 754–756, illus.
(6) BAILEY, F. D.
1913. ONION DISEASES. Oregon Agr. Expt. Sta. Crop Pest and Hort. Bien. Rpt. 1911–12: 276.
(7) BARKER, B. T. P., and GIMINGHAM, C. T.
[1917.] EXPERIMENTS ON THE TREATMENT OF RHIZOCTONIA DISEASE OF ASPARAGUS. Bristol Univ., Agr. and Hort. Res. Sta. Ann. Rept. 1916: 39–40.
(8) BISSON, C. S., JONES, H. A., and ROBBINS, W. W.
1926. FACTORS INFLUENCING THE QUALITY OF FRESH ASPARAGUS AFTER IT IS HARVESTED. Calif. Agr. Expt. Sta. Bul. 410, 28 pp., illus.
(9) BODINE, E. W., and DURRELL, L. W.
1931. PURPLE BLOTCH OF ONIONS. Colo. Agr. Col. Ext. Cir. 1194–A, [4] pp., illus.
(10) COOK, HAROLD THURSTON.
1932. STUDIES ON DOWNY MILDEW OF ONIONS, AND THE CAUSAL ORGANISM, PERONOSPORA DESTRUCTOR (BERK.) CASPARY. N. Y. (Cornell) Agr. Expt. Sta. Mem. 143, 40 pp., illus.
(11) COOK, MEL T.
1922. A NEW DISEASE OF ASPARAGUS. (Abstract) Phytopathology 12: 49.
(12) COTTON, A. D., and OWEN, M. N.
1920. THE WHITE ROT DISEASE OF ONION BULBS. [Gt. Brit.] Min. Agr. and Fisheries Jour. 26: 1093–1099, illus.
(13) DAVIS, GLEN N., and HENDERSON, W. J.
1937. THE INTERRELATION OF THE PATHOGENICITY OF A PHOMA AND A FUSARIUM ON ONIONS. Phytopathology 27: 763–772, illus.
(14) ——— and REDDY, C. H.
1932. A SEEDLING-BLIGHT STAGE OF ONION BULB ROT. (Abstract) Phytopathology 22: 8.
(15) HANSEN, H. N.
1929. ETIOLOGY OF THE PINK-ROOT DISEASE OF ONIONS. Phytopathology 19: 691–704, illus.
(16) HENDERSON, W. H.
1930. INDEXING AS A CONTROL MEASURE FOR THE YELLOW-DWARF DISEASE OF ONIONS. (Abstract) Phytopathology 20: 115.

(17) LINK, GEORGE K. K., and BAILEY, ALICE A.
1926. FUSARIA CAUSING BULB ROT OF ONIONS. Jour. Agr. Res. 33: 929–952, illus.
(18) ——— and GARDNER, MAX W.
1919. MARKET PATHOLOGY AND MARKET DISEASES OF VEGETABLES. Phytopathology 9: [497]–520.
(19) LUTZ, J. M.
1936. THE INFLUENCE OF RATE OF THAWING ON FREEZING INJURY OF APPLES, POTATOES AND ONIONS. Amer. Soc. Hort. Sci. Proc. (1935) 33: 227–233, illus.
(20) MACHACEK, J. E.
1929. THE BLACK MOLD OF ONIONS, CAUSED BY ASPERGILLUS NIGER V. TIEGH. Phytopathology 19: 733–739, illus.
(21) MUNN, M. T.
1917. NECK-ROT DISEASE OF ONIONS. N. Y. State Agr. Expt. Sta. Bul. 437, pp. 361–455, illus.
(22) MURPHY, PAUL A.
1921. THE PRESENCE OF PERENNIAL MYCELIUM IN PERONOSPORA SCHLEIDENI, UNGER. Nature [London] 108: 304.
(23) NEWHALL, A. G.
1938. THE SPREAD OF ONION MILDEW BY WIND-BORNE CONIDIA OF PERONOSPORA DESTRUCTOR. Phytopathology 28: 257–269, illus.
(24) NORTON, J. B.
1913. METHODS USED IN BREEDING ASPARAGUS FOR RUST RESISTANCE. U. S. Dept. Agr., Bur. Plant Indus. Bul. 263, 60 pp., illus.
(25) PENTZER, W. T., et al.
1936. PRECOOLING AND SHIPPING CALIFORNIA ASPARAGUS. Calif. Agr. Expt. Sta. Bul. 600, 45 pp. illus.
(26) PORTER, D. R., and JONES, H. A.
1933. RESISTANCE OF SOME OF THE CULTIVATED SPECIES OF ALLIUM TO PINK ROOT (PHOMA TERRESTRIS). Phytopathology 23: 290–298, illus.
(27) RAMSEY, G. B.
1930. BLEMISHES AND DISCOLORATIONS OF MARKET ONIONS. U. S. Dept. Agr. Cir. 135, 4 pp., illus.
(28) ——— and BUTLER, L. F.
1928. INJURY TO ONIONS AND FRUITS CAUSED BY EXPOSURE TO AMMONIA. Jour. Agr. Res. 37: 339–348, illus.
(29) RIEMAN, G. H.
1931. GENETIC FACTORS FOR PIGMENTATION IN THE ONION AND THEIR RELATION TO DISEASE RESISTANCE. Jour. Agr. Res. 42: 251–278, illus.
(30) SIDERIS, CHRISTOS P.
1924. SPECIES OF FUSARIUM ISOLATED FROM ONION ROOTS. Phytopathology 14: [211]–216, illus.
(31) SMITH, RALPH. E.
1905. ASPARAGUS AND ASPARAGUS RUST IN CALIFORNIA. Calif. Agr. Expt. Sta. Bul. 165, 99 pp., illus.
(32) ———
1906. FURTHER EXPERIENCE IN ASPARAGUS RUST CONTROL. Calif. Agr. Expt. Sta. Bul. 172, 21 pp., illus.
(33) STEVENS, FRANK LINCOLN, and TRUE, ESTHER YOUNG.
[1919.] BLACK SPOT OF ONION SETS. Ill. Agr. Expt. Sta. Bul. 220, pp. 507–532, illus.
(34) STEWART, F. C.
1899. I. A. BACTERIAL ROT OF ONIONS. *In* Notes on Various Plant Diseases. N. Y. State Agr. Expt. Sta. Bul. 164: [209]–212, illus.
(35) STONE, G. E., and SMITH, R. E.
1899. THE ASPARAGUS RUST IN MASSACHUSETTS. Mass. Agr. Expt. Sta. Bul. 61, 20 pp., illus.
(36) TAUBENHAUS, J. J., and EZEKIEL, WALTER N.
1934. ALKALI SCORCH OF BERMUDA ONIONS. Amer. Jour. Bot. 21: 69–71, illus.
(37) ——— and MALLY, FREDERICK W.
1921. PINK ROOT DISEASE OF ONIONS AND ITS CONTROL IN TEXAS. Tex. Agr. Expt. Sta. Bul. 273, 42 pp., illus.

(38) THAXTER, ROLAND.
1890. THE ONION MACROSPORIUM (MACROSPORIUM SARCINULA BERK., VARIETY PARASITICUM THÜM.). Conn. (State) Agr. Expt. Sta. Ann. Rpt. 1889: 158–161, illus.
(39) ———
1890. THE "SMUT" OF ONIONS, (UROCYSTIS CEPULAE FROST.). Conn. (State) Agr. Expt. Sta. Ann. Rpt. 1889: 129–153, illus.
(40) VAN PELT, WAYNE.
1917. BLACK MOLD OF ONIONS, A NEW FUNGUS DISEASE CAUSING SERIOUS DAMAGE IN STORAGE HOUSES. Ohio Agr. Expt. Sta. Monthly Bul. 2: 152–156, illus.
(41) WALKER, J. C.
1918. CONTROL OF NECK ROT AND ANTHRACNOSE OF ONION SETS. Phytopathology 8: 70.
(42) ———
1920. EXPERIMENTS UPON FORMALDEHYDE-DRIP CONTROL OF ONION SMUT. Phytopathology 10: [323]–327.
(43) ———
1921. RUST OF ONION FOLLOWED BY A SECONDARY PARASITE. Phytopathology 11: 87–90, illus.
(44) ———
1921. A MACROSPORIUM ROT OF ONION. (Abstract) Phytopathology 11: 53.
(45) ———
1921. ONION SMUDGE. Jour. Agr. Res. 20: 685–722, illus.
(46) ———
1923. DISEASE RESISTANCE TO ONION SMUDGE. Jour. Agr. Res. 24: 1019–1040, illus.
(47) ———
1924. WHITE ROT OF ALLIUM IN EUROPE AND AMERICA. Phytopathology 14: [315]–322, illus.
(48) ———
1925. CONTROL OF MYCELIAL NECK ROT OF ONION BY ARTIFICIAL CURING. Jour. Agr. Res. 30: 365–373, illus.
(49) ———
1926. BOTRYTIS NECK ROTS OF ONIONS. Jour. Agr. Res. 33: 893–928, illus.
(50) ———
1931. ONION DISEASES AND THEIR CONTROL. U. S. Dept. Agr. Farmers' Bul. 1060, 25 pp., illus. [Revised.]
(51) ——— and JONES, L. R.
1921. RELATION OF SOIL TEMPERATURE AND OTHER FACTORS TO ONION SMUT INFECTION. Jour. Agr. Res. 22: 235–262, illus.
(52) ——— and LINDEGREN, CARL C.
1924. FURTHER STUDIES ON THE RELATION OF ONION SCALE PIGMENTATION TO DISEASE RESISTANCE. Jour. Agr. Res. 29: 507–514.
(53) ——— and LINK, KARL PAUL.
1935. TOXICITY OF PHENOLIC COMPOUNDS TO CERTAIN ONION BULB PARASITES. Bot. Gaz. 96: 468–484.
(54) ——— and MURPHY, ALBERT.
1934. ONION-BULB DECAY CAUSED BY ASPERGILLUS ALLIACEUS. Phytopathology 24: 289–291, illus.
(55) ——— and TIMS, E. C.
1924. A FUSARIUM BULB ROT OF ONION AND THE RELATION OF ENVIRONMENT TO ITS DEVELOPMENT. Jour. Agr. Res. 28: 683–694, illus.
(56) WOLLENWEBER, H. W., and REINKING, O. A.
1935. DIE FUSARIEN, IHRE BESCHRIEBUNG, SCHADWIRKUNG UND BEKÄMPFUNG. 355 pp., illus. Berlin.
(57) WRIGHT, R. C.
1927. SOME EFFECTS OF FREEZING ON ONIONS. U. S. Dept. Agr. Dept. Cir. 415, 8 pp., illus.
(58) ——— LAURITZEN, J. I., and WHITEMAN, T. M.
1935. INFLUENCE OF STORAGE TEMPERATURE AND HUMIDITY ON KEEPING QUALITIES OF ONIONS AND ONION SETS. U. S. Dept. Agr. Tech. Bul. 475, 38 pp., illus.

Pulse Family

(59) Anderson, H. W.
1924. Notes of the nematospora disease of lima beans. (Abstract) Phytopathology 14: 31.
(60) Andrus, C. F.
1938. Seed transmission of macrophomina phaseoli. Phytopathology 28: 620–634, illus.
(61) ——— and Moore, W. D.
1935. Colletotrichum truncatum (Schw.), n. comb., on garden and lima beans. Phytopathology 25: 121–125, illus.
(62) Barrus, Mortimer F.
1910. Rhizoctonia stem rot of beans. Science (n. s.) 31: 796–797.
(63) ———
1918. Varietal susceptibility of beans to strains of colletotrichum lindemuthianum (Sacc. & Magn.) B. & C. Phytopathology 8: [589]–614, illus.
(64) ———
1921. Bean anthracnose. N. Y. (Cornell) Agr. Expt. Sta. Mem. 42: [95]–213, illus.
(65) Beach, W. S.
1928. The relation of bacterium vignae to the tissues of lima bean. Pa. Agr. Expt. Sta. Bul. 226, 15 pp., illus.
(66) Bisby, G. R.
1918. A fusarium disease of garden peas in Minnesota. (Abstract) Phytopathology 8: 77.
(67) Brooks, Charles, and McColloch, L.
1938. Stickiness and spotting of shelled green lima beans. U. S. Dept. Agr. Tech. Bul. 625, 24 pp., illus.
(68) Bruner, S. C., and Arango, Oscar.
1931. La enfermedad "verruga" de las habas de lima. Cuba Estac. Expt. Agron. Cir. 74, 38 pp., illus.
(69) ——— and Jenkins, Anna E.
1933. Identity and host relations of the elsinoe of lima bean. Jour. Agr. Res. 47: 783–789, illus.
(70) Burkholder, Walter H.
1919. The dry root rot of the bean. N. Y. (Cornell) Agr. Expt. Sta. Mem. 26: 1003–1033, illus.
(71) ———
1921. The bacterial blight of the bean: a systemic disease. Phytopathology 11: [61]–69.
(72) ———
1923. The gamma strain of colletotrichum lindemuthianum (Sacc. et Magn.) B. et C. Phytopathology 13: 316–323.
(73) ———
1924. The effct of varying soil moistures on healthy bean plants and on those infected by a root parasite. Ecology 5: 179–187.
(74) ———
1924. Varietal susceptibility among beans to the bacterial blight. Phytopathology 14: [1]–7.
(75) ———
1926. A new bacterial disease of the bean. Phytopathology 16: 915–927, illus.
(76) ———
1930. The bacterial diseases of the bean, a comparative study. N. Y. (Cornell) Agr. Expt. Sta. Mem. 127, 88 pp., illus.
(77) ——— and Zaleski, Karol.
1932. Varietal susceptibility of beans to an American and a European strain of phytomonas medicaginis var. phaseolicola, and a comparison of the strains in culture. Phytopathology 22: 85–94.
(78) Campbell, Leo.
1935. Downy mildew of peas caused by peronospora pisi (de B.) Syd. Wash. Agr. Expt. Sta. Bul. 318, 42 pp., illus.

(79) CLINTON, G. P.
1906. DOWNY MILDEW, PHYTOPTHORA PHASEOLI THAXT., OF LIMA BEANS. Conn. (State) Agr. Expt. Sta. Ann. Rpt. 1905: 278–303.
(80) COOK, HAROLD T.
1931. POWDERY MILDEW DISEASE OF SNAP BEANS. Va. Truck Expt. Sta. Bul. 74: [929]–940, illus.
(81) COONS, G. H.
1919. MICHIGAN EXPERIMENTS ON BEAN DISEASE CONTROL. Mich. Agr. Expt. Sta. Quart. Bul. 1: 104–106, illus.
(82) DICKSON, B. T.
1922. STUDIES CONCERNING MOSAIC DISEASES. MacDonald Col., McGill Univ., Tech. Bul. 2, 125 pp., illus.
(83) DRECHSLER, CHARLES.
1925. ROOT-ROT OF PEAS IN THE MIDDLE ATLANTIC STATES IN 1924. Phytopathology 15: [110]–114.
(84) EDGERTON, C. W.
1910. THE BEAN ANTHRACNOSE. La. Agr. Sta. Bul. 119, 55 pp., illus.
(85) ——— and MORELAND, C. C.
1913. THE BEAN BLIGHT AND PRESERVATION AND TREATMENT OF BEAN SEED. La. Agr. Expt. Sta. Bul. 139, 43 pp., illus.
(86) FAJARDO, T. G.
1930. STUDIES ON THE MOSAIC DISEASE OF THE BEAN (PHASEOLUS VULGARIS L.). Phytonathology 20: 469–494, illus.
(87) FROMME, F. D., and WINGARD, S. A.
1918. BEAN RUST. Va. Agr. Expt. Sta. Bul. 220, 18 pp., illus.
(88) ——— and WINGARD, S. A.
1921. VARIETAL SUSCEPTIBILITY OF BEANS TO RUST. Jour. Agr. Res. 21: 385–404, illus.
(89) GALLOWAY, B. T.
1889. POWDERY MILDEW OF THE BEAN. Jour. Mycol 5: 214.
(90) GARDNER, MAX W., and KENDRICK, JAMES B.
1923. BACTERIAL SPOT OF COWPEA. Science (n. s.) 57: 275.
(91) ——— and KENDRICK, J. B.
1925. BACTERIAL SPOT OF COWPEA AND LIMA BEAN. Jour. Agr. Res. 31: 841–863, illus.
(92) GLOYER, W. O.
1924. THE EFFECT OF LATE PLANTING ON THE BACTERIAL BLIGHT OF BEANS. (Abstract) Phytopathology 14: 27.
(93) HALSTED, BYRON D.
1901. BEAN DISEASES AND THEIR REMEDIES. N. J. Agr. Expt. Sta. Bul. 151, 28 pp. illus.
(94) ———
1901. THE DOWNY MILDEW OF LIMA BEANS. N. J. Agr. Expt. Sta. Bul. 151: 18–24, illus.
(95) HARTER, L. L.
1917. PODBLIGHT OF THE LIMA BEAN CAUSED BY DIAPORTHE PHASEOLORUM. Jour. Agr. Res. 11: 473–504, illus.
(96) ———
1938. A ROOT ROT OF PEAS CAUSED BY FUSARIUM COERULEUM. Phytopathology 28: 432–438, illus.
(97) ——— ANDRUS, C. F., and ZAUMEYER, W. J.
1935. STUDIES ON BEAN RUST CAUSED BY UROMYCES PHASEOLI TYPICA. Jour. Agr. Res. 50: 737–758, illus.
(98) ——— and WHITNEY, W. A.
1927. A TRANSIT DISEASE OF SNAP BEANS CAUSED BY PYTHIUM APHANIDERMATUM. Jour. Agr. Res. 34: 443–447, illus.
(99) ——— and ZAUMEYER, W. J.
1931. PYTHIUM BUTLERI—THE CAUSE OF A BEAN WILT. Phytopathology 21: 991–994.
(100) ——— and ZAUMEYER, W. J.
1932. BEAN DISEASES AND THEIR CONTROL. U. S. Dept. Agr. Farmers' Bul 1692, 28 pp., illus.
(101) ——— ZAUMEYER, W. J., and WADE, B. L.
1934. PEA DISEASES AND THEIR CONTROL. U. S. Dept. Agr. Farmers' Bul. 1735, 25 pp., illus.

(102) HEDGES, FLORENCE.
1922. A BACTERIAL WILT OF THE BEAN CAUSED BY BACTERIUM FLACCUMFACIENS, NOV. SP. (N. S.). Science (n. s.) 55: 433–434.
(103) ———
1926. BACTERIAL WILT OF BEANS (BACTERIUM FLACCUMFACIENS HEDGES), INCLUDING COMPARISONS WITH BACTERIUM PHASEOLI. Phytopathology 16: [1]–22, illus.
(104) JENKINS, ANNA E.
1931. LIMA-BEAN SCAB CAUSED BY ELSINOE. Jour. Agr. Res. 42: 13–23, illus.
(105) ———
1933. FURTHER STUDIES OF LIMA-BEAN SCAB. Phytopathology 23: 662–666, illus.
(106) JENNISON, HARRY MILLIKEN.
1921. OBSERVATIONS UPON THE BACTERIAL BLIGHT OF FIELD AND GARDEN PEAS IN MONTANA. Phytopathology 11: 104.
(107) JOHNSON, JAMES.
1916. HOST PLANTS OF THIELAVIA BASICOLA. Jour. Agr. Res. 7: 289–300, illus.
(108) ——— and HARTMAN, R. E.
1918. INFLUENCE OF SOIL TEMPERATURE ON THIELAVIA ROOT-ROT. (Abstract) Phytopathology 8: 77.
(109) JONES, FRED REUEL.
1923. STEM AND ROOTROT OF PEAS IN THE UNITED STATES CAUSED BY SPECIES OF FUSARIUM. Jour. Agr. Res. 26: 459–476, illus.
(110) ——— and DRECHSLER, CHARLES.
1925. ROOTROT OF PEAS IN THE UNITED STATES CAUSED BY APHANOMYCES EUTEICHES (N. SP.). Jour. Agr. Res. 30: 293–325, illus.
(111) ——— and LINFORD, M. B.
1925. PEA DISEASE SURVEY IN WISCONSIN. Wis. Agr. Expt. Sta. Res. Bul. 64, 30 pp., illus.
(112) ——— and VAUGHAN, R. E.
1921. ANTHRACNOSE OF THE GARDEN PEA. Phytopathology 11: 500–503, illus.
(113) JONES, LEON K.
1927. STUDIES OF THE NATURE AND CONTROL OF BLIGHT, LEAF AND POD SPOT AND ROOTROT OF PEAS CAUSED BY SPECIES OF ASCOCHYTA. N. Y. State Agr. Expt. Sta. Bul. 547, 46 pp., illus.
(114) KENDRICK, JAMES B.
1933. SEEDLING STEM BLIGHT OF FIELD BEANS CAUSED BY RHIZOCTONIA BATATICOLA AT HIGH TEMPERATURES. Phytopathology 23: 949–963, illus.
(115) Number not used.
(116) LAURITZEN, J. I.
1919. THE RELATION OF TEMPERATURE AND HUMIDITY TO INFECTION BY CERTAIN FUNGI. Phytopathology 9: [7]–35.
(117) ——— HARTER, L. L., and WHITNEY, W. A.
1933. ENVIRONMENTAL FACTORS IN RELATION TO SNAP-BEAN DISEASES OCCURRING IN SHIPMENT. Phytopathology 23: 411–445, illus.
(118) LEACH, J. G.
1923. THE PARASITISM OF COLLECTOTRICHUM LINDEMUTHIANUM. Minn. Agr. Expt. Sta. Tech. Bul. 14, 42 pp., illus.
(119) LEONARD, LEWIS T.
1923. AN INFLUENCE OF MOISTURE ON BEAN WILT. Jour. Agr. Res. 24: 749–752, illus.
(120) LINFORD, M[AURICE] B.
1931. STREAK, A VIRUS DISEASE OF PEAS TRANSMITTED BY THRIPS TABACI. (Abstract) Phytopathology 21: 999.
(121) ——— and SPRAGUE, RODERICK.
1927. SPECIES OF ASCOCHYTA PARASITIC ON THE PEA. Phytopathology 17: 381–397, illus.
(122) LUDWIG, C. A.
1926. PSEUDOMONAS (PHYTOMONAS) PISI SACKETT, THE CAUSE OF A POD SPOT OF GARDEN PEAS. Phytopathology 16: 177–183, illus.

(123) MACKIE, W. W.
1932. A HITHERTO UNREPORTED DISEASE OF MAIZE AND BEANS. Phytopathology 22: 637–644, illus.
(124) MACMILLAN, H. G.
1918. SUNSCALD OF BEANS. Jour. Agr. Res. 13: 647–650, illus.
(125) ——— and BYARS, L. P.
1920. HEAT INJURY TO BEANS IN COLORADO. Phytopathology 10: [365]–367, illus.
(126) ———
1923. CAUSE OF SUNSCALD OF BEANS. Phytopathology 13: [376]–380.
(127) MCCLINTOCK, J. A.
1916. SCLEROTINIA LIBERTIANA ON SNAP BEANS. Phytopathology 6: [436]–441, illus.
(128) ———
1916. SCLEROTINIA BLIGHT, A SERIOUS DISEASE OF SNAP BEANS CAUSED BY SCLEROTINIA LIBERTIANA, FCKL. Va. Truck Expt. Sta. Bul. 20, pp. 419–428, illus.
(129) MOORE, W. D.
1936. POWDERY MILDEW (ERYSIPHE POLYGONI) ON GARDEN SNAP BEANS. Phytopathology 26: 1135–1144, illus.
(130) MUNCIE, J. H.
1914. TWO MICHIGAN BEAN DISEASES. Mich. Agr. Sta. Special Bul. 68, 12 pp., illus.
(131) ———
1917. EXPERIMENTS ON THE CONTROL OF BEAN ANTHRACNOSE AND BEAN BLIGHT. Mich. Agr. Expt. Sta. Tech. Bul. 38, 50 pp., illus.
(132) MURPHY, DONALD M., and PIERCE, W. H.
1937. COMMON MOSAIC OF THE GARDEN PEA, PISUM SATIVUM. Phytopathology 27: 710–721, illus.
(132a) PAINE, SYDNEY G., and LACEY, MARGARET S.
1922. CHOCOLATE SPOT DISEASE OR STREAK DISEASE OF BROAD BEANS. [Gt. Brit.] Min. Agr. and Fisheries Jour. 29: 175–177, illus.
(133) PIERCE, WALTER H.
1934. VIROSES OF THE BEAN. Phytopathology 24: 87–115, illus.
(134) RANDS, R. D., and BROTHERTON, WILBUR.
1925. BEAN VARIETAL TESTS FOR DISEASE RESISTANCE. Jour. Agr. Res. 31: 101–154, illus.
(135) RAPP, C. W.
1920. BACTERIAL BLIGHT OF BEANS, A TECHNICAL STUDY. Okla. Agr. Expt. Sta. Bul. 131, 39 pp., illus.
(136) REDDICK, DONALD.
1917. EFFECT OF SOIL TEMPERATURE ON THE GROWTH OF BEAN PLANTS AND ON THEIR SUSCEPTIBILITY TO A ROOT PARASITE. Amer. Jour. Bot. 4: 513–519.
(137) ———
1922. A HYBRID BEAN RESISTANT TO ANTHRACNOSE AND TO MOSAIC. (Abstract) Phytopathology 12: 47.
(138) ——— and STEWART, VERN B.
1918. VARIETIES OF BEANS SUSCEPTIBLE TO MOSAIC. Phytopathology 8: [530]–534.
(139) ——— and STEWART, VERN B.
1919. TRANSMISSION OF THE VIRUS OF BEAN MOSAIC IN SEED AND OBSERVATIONS ON THERMAL DEATH-POINT OF SEED AND VIRUS. Phytopathology 9: [445]–450.
(140) RICHARDS, B. L.
1923. SOIL TEMPERATURE AS A FACTOR AFFECTING THE PATHOGENICITY OF CORTICIUM VAGUM ON THE PEA AND THE BEAN. Jour. Agr. Res. 25: 431–450, illus.
(141) SACKETT, WALTER G.
1916. A BACTERIAL STEM BLIGHT OF FIELD AND GARDEN PEAS. Colo. Agr. Expt. Sta. Bul. 218, 43 pp., illus.
(142) SKORIC, VLADIMIR.
1927. BACTERIAL BLIGHT OF PEA: OVERWINTERING, DISSEMINATION, AND PATHOLOGICAL HISTOLOGY. Phytopathology 17: 611–627, illus.

(143) SNYDER, WILLIAM C.
1934. POD DEFORMATION OF MOSAIC-INFECTED PEAS. (Phytopath. note.) Phytopathology 24: 78–80, illus.
(144) ———
1934. A LEAF, STEM, AND POD SPOT OF PEA CAUSED BY A SPECIES OF CLADOSPORIUM. Phytopathology 24: 890–905, illus.
(145) ———
1934. PERONOSPORA VICIAE AND INTERNAL PROLIFERATION IN PEA PODS. Phytopathology 24: 1358–1365, illus.
(146) STUBBS, MERLE W.
1937. CERTAIN VIRUSES OF THE GARDEN PEA, PISUM SATIVUM. Phytopathology 27: 242–266, illus.
(147) TISDALE, W. B., and WILLIAMSON, MAUDE MILLER.
1923. BACTERIAL SPOT OF LIMA BEAN. Jour. Agr. Res. 25: 141–154, illus.
(148) VAN HOOK, J. M.
1906. BLIGHTING OF FIELD AND GARDEN PEAS. Ohio Agr. Expt. Sta. Bul. 173, pp. 231–249, illus.
(149) VAUGHAN, R. E.
1913. MYCOSPHAERELLA PINODES THE ASCIGEROUS STAGE OF ASCOCHYTA PISI. (Abstract) Phytopathology 3: 71–72.
(150) ———
1916. THE DEVELOPMENT OF MYCOSPHAERELLA PINODES IN PURE CULTURE. (Abstract) Phytopathology 6: 103.
(151) WALKER, J. C., and SNYDER, W. C.
1933. PEA WILT AND ROOT ROTS. Wis. Agr. Expt. Sta. Bul. 424, 16 pp., illus.
(152) WHETZEL, H. H.
1908. BEAN ANTHRACNOSE. N. Y. (Cornell) Agr. Expt. Sta. Bul. 255, pp. [429]–447, illus.
(153) WHIPPLE, OTIS C.
1936. SPOTTED WILT OF GARDEN PEA. (Phytopath. note.) Phytopathology 26: 918–929, illus.
(154) WINGARD, S. A.
1922. YEAST-SPOT OF LIMA BEANS. Phytopathology 12: [525]–532, illus.
(155) ZAUMEYER, W. J.
1930. THE BACTERIAL BLIGHT OF BEANS CAUSED BY BACTERIUM PHASEOLI. U. S. Dept. Agr. Tech. Bull. 186, 36 pp., illus.
(156) ———
1932. COMPARATIVE PATHOLOGICAL HISTOLOGY OF THREE BACTERIAL DISEASES OF BEAN. Jour. Agr. Res. 44: 605–632, illus.
(157) ——— and KEARNS, C. W.
1936. THE RELATION OF APHIDS TO THE TRANSMISSION OF BEAN MOSAIC. Phytopathology 26: 614–629.
(158) ——— and WADE, B. L.
1933. MOSAIC DISEASES AFFECTING DIFFERENT LEGUMES IN RELATION TO BEANS AND PEAS. (Phytopath. note.) Phytopathology 23: 562–564.
(159) ——— and WADE, B. L.
1934. PHYSIOLOGICAL SPOTTING OF PEA SEED. (Phytopath. note.) Phytopathology 24: 1383–1384, illus.

PARSLEY FAMILY

(160) [BEACH, S. A.]
1893. SOME CELERY DISEASES. N. Y. State Agr. Expt. Sta. Bul. 51, pp. [133]–148, illus.
(161) BEATTIE, W. R.
1936. CELERY GROWING. U. S. Dept. Agr. Farmers' Bul. 1269, 46 pp., illus. [Revised.]
(162) BENNETT, C. W.
1921. A PHOMA ROOT ROT OF CELERY. Mich. Agr. Expt. Sta. Tech. Bul. 53, 40 pp., illus.
(163) CHUPP, CHARLES.
1925. MANUAL OF VEGETABLE-GARDEN DISEASES. 647 pp., illus. New York.
(164) COCHRAN, L. C.
1932. A STUDY OF TWO SEPTORIA LEAF SPOTS OF CELERY. Phytopathology 22: 791–812, illus.

(165) COTTON, A. D.
1918. DISEASES OF PARSNIP. STUDIES FROM THE PATHOLOGICAL LABORATORY: VI. Kew Roy. Bot. Gard. Bul. Misc. Inform. 1918: 8–21, illus.
(166) CROSBY, C. R., and CHUPP, CHARLES.
1931. THE CONTROL OF DISEASES AND INSECTS AFFECTING VEGETABLE CROPS. N. Y. Agr. Col. (Cornell) Ext. Bul. 206, 101 pp., illus.
(167) ——— and LEONARD, M. D.
1914. THE TARNISHED PLANT-BUG. N. Y. (Cornell) Agr. Expt. Sta. Bul. 346, pp. [461]–526, illus.
(168) DOOLITTLE, S. P., and WELLMAN, F. L.
1934. COMMELINA NUDIFLORA, A MONOCOTYLEDONOUS HOST OF A CELERY MOSAIC IN FLORIDA. Phytopathology 24: 48–61, illus.
(169) DORAN, W. L., and GUBA, E. F.
1928. BLIGHT AND LEAF-SPOT OF CARROT IN MASSACHUSETTS. Mass. Agr. Expt. Sta. Bul. 245, pp. [269]–278.
(170) DYE, H. W., and NEWHALL, A. G.
1924. THE CONTROL OF BACTERIAL BLIGHT OF CELERY BY SPRAYING AND DUSTING. N. Y. (Cornell) Agr. Expt. Sta. Bul. 429, 30 pp., illus. [Revised 1925.]
(171) EMSWELLER, S. L.
1933. AN HEREDITARY PITHINESS IN CELERY. Amer. Soc. Hort. Sci. Proc. (1932) 29: 480–485, illus.
(172) FOSTER, ARTHUR C.
1927. CELERY DISEASE AND ITS CONTROL. U. S. Dept. Agr. Yearbook 1926: 222–223.
(173) ——— and WEBER, G. F.
1924. CELERY DISEASES IN FLORIDA. Fla. Agr. Expt. Sta. Bul. 173, pp. [21]–77, illus.
(174) GARDNER, M. W., TOMPKINS, C. M., and THOMAS, H. REX.
1937. FACTORS AFFECTING THE PREVALENCE OF THE SPOTTED WILT VIRUS. (Abstract) Phytopathology 27: 129.
(175) GONZALEZ, L. G.
[1927.] SOME FREEZING STUDIES ON CELERY. Amer. Soc. Hort. Sci. Proc. (1926) 23: 339–351.
(176) GOOSSENS, J. A. A. M. H.
1928. ONDERZOEK OVER DE DOOR PHOMA APIICOLA KLEBAHN VEROORZAAKTE SCHURFTZIEKTE VAN DE KNOLSELDERIJ EN OVER SYNERGETISCHE VORMEN EN LOCALE RASSEN VAN DEZE ZWAM. Tijdschr. over Plantenziekten 34: 271–348, illus. [In Dutch. English summary, pp. [343]–344.]
(177) HARDING, H. A., MORSE, W. J., and JONES, L. R.
1909. THE BACTERIAL SOFT ROTS OF CERTAIN VEGETABLES. N. Y. State Agr. Expt. Sta. Tech. Bul. 11, pp. (251)–364, illus. [Also Vt. Agr. Expt. Sta. Bul. 147, 1910.]
(178) JAGGER, IVAN C.
1920. SCLEROTINIA MINOR, N. SP., THE CAUSE OF A DECAY OF LETTUCE, CELERY, AND OTHER CROPS. Jour. Agr. Res. 20: 331–334, illus.
(179) ———
1921. BACTERIAL LEAFSPOT DISEASE OF CELERY. Jour. Agr. Res. 21: 185–188, illus.
(180) JONES, L. R.
1901. A SOFT ROT OF CARROT AND OTHER VEGETABLES CAUSED BY BACILLUS CAROTOVORUS, JONES. Vt. Agr. Expt. Sta. Ann. Rpt. (1899–1900) 13: 299–332, illus.
(181) KENDRICK, JAMES B.
1934. BACTERIAL BLIGHT OF CARROT. Jour. Agr. Res. 49: 493–510, illus.
(182) KLOTZ, L. J.
1923. A STUDY OF THE EARLY BLIGHT FUNGUS, CERCOSPORA APII FRES. Mich. Agr. Expt. Sta. Tech. Bul. 63, 43 pp., illus.
(183) KROUT, WEBSTER S.
1921. TREATMENT OF CELERY SEED FOR THE CONTROL OF SEPTORIA BLIGHT. Jour. Agr. Res. 21: 369–372.
(184) KUNKEL, L. O.
1926. STUDIES ON ASTER YELLOWS. Boyce Thompson Inst. Contrib. 1: 181–240, illus.

(185) KUNKEL, L. O.
1931. STUDIES ON ASTER YELLOWS IN SOME NEW HOST PLANTS. Boyce Thompson Inst. Contrib. 3: 85–123, illus.
(186) ———
1932. CELERY YELLOWS OF CALIFORNIA NOT IDENTICAL WITH THE ASTER YELLOWS OF NEW YORK. Boyce Thompson Inst. Contrib. 4: 405–414, illus.
(187) LAURITZEN, J. I.
1926. THE RELATION OF BLACK ROT TO THE STORAGE OF CARROTS. Jour. Agr. Res. 33: 1025–1041, illus.
(188) ———
1932. DEVELOPMENT OF CERTAIN STORAGE AND TRANSIT DISEASES OF CARROT. Jour. Agr. Res. 44: 861–912, illus.
(189) LEACH, J. G.
1927. THE RELATION OF INSECTS AND WEATHER TO THE DEVELOPMENT OF HEART ROT OF CELERY. Phytopathology 17: 663–667, illus.
(190) MCCLINTOCK, J. A.
1916. A DISEASE OF COLDFRAME PARSLEY CAUSED BY SCLEROTINIA LIBERTIANA. Va. Truck Expt. Sta. Bul. 18, pp. 379–384, illus.
(191) MEIER, FRED C., DRECHSLER, CHARLES, and EDDY, E. D.
1922. BLACK ROT OF CARROTS CAUSED BY ALTERNARIA RADICINA, N. SP. Phytopathology 12: [157]–166, illus.
(192) MICHIGAN AGRICULTURAL EXPERIMENT STATION.
1938. CELERY PRODUCTION IN MICHIGAN. Mich. Agr. Expt. Sta. Cir. Bul. 165, 43 pp., illus.
(193) NELSON, RAY COONS, G. H., and COCHRAN, L. C.
1937. THE FUSARIUM YELLOWS DISEASE OF CELERY (APIUM GRAVEOLENS L. VAR DULCE D. C.). Mich. Agr. Expt. Sta. Tech. Bul. 155, 74 pp., illus.
(194) NEWHALL, A. G.
1926. THE IMPORTANCE OF CONTROLLING CELERY BLIGHT IN THE SEEDBED. Phytopathology 16: 467–472, illus.
(195) ———
1932. ROOT AND STORAGE ROT OF CELERY CAUSED BY PHOMA APIICOLA. (Abstract) Phytopathology 22: 21.
(196) POOLE, R. F[rank].
1922. CELERY MOSAIC. Phytopathology 12: [151]–154, illus.
(197) ———
1922. SOME RECENT INVESTIGATIONS ON THE CONTROL OF SCLEROTINIA LIBERTIANA IN THE GREENHOUSE ON THE MUCK FARMS OF BERGEN COUNTY, NEW JERSEY. Phytopathology 12: 16–20, illus.
(198) ———
1922. THE SCLEROTINIA ROT OF CELERY. N. J. Agr. Expt. Sta. Bul. 359, 27 pp., illus.
(199) PRICE, W. C.
1935. CLASSIFICATION OF SOUTHERN CELERY-MOSAIC VIRUS. Phytopathology 25: 947–954, illus.
(200) PURVIS, E. R., and RUPRECHT, R. W.
1935. BORAX AS A FERTILIZER FOR CELERY. Fla. Agr. Expt. Sta. Press Bul. 478, [2] pp.
(201) ——— and RUPRECHT, R. W.
1937. CRACKED STEM OF CELERY CAUSED BY A BORON DEFICIENCY IN THE SOIL. Fla. Agr. Expt. Sta. Bul. 307, 16 pp., illus.
(202) RAMSEY, G. B.
1924. SCLEROTINIA INTERMEDIA N. SP. A CAUSE OF DECAY OF SALSIFY AND CARROTS. Phytopathology 14 [323]–327, illus.
(203) ———
1925. SCLEROTINIA SPECIES CAUSING DECAY OF VEGETABLES UNDER TRANSIT AND MARKET CONDITIONS. Jour. Agr. Res. 31: 597–632, illus.
(204) REDDICK, D.
1914. DECAY OF CELERY IN STORAGE. (Abstract) Phytopathology 4: 45.
(205) RICHARDSON, J. K.
1936. CONTROL OF LATE BLIGHT OF CELERY. Sci. Agr. 16: 358–364, illus.
(206) ———
1938. STUDIES ON BLACKHEART, SOFT-ROT, AND TARNISHED PLANT BUG INJURY OF CELERY. Canad. Jour. Res., Sect. C, Bot. Sci. 16: 182–193, illus.

(207) ROGERS, STANLEY S.
1911. THE LATE BLIGHT OF CELERY. Calif. Agr. Expt. Sta. Bul. 208, pp. [81]–115, illus.
(208) SEVERIN, HENRY H. P.
1929. YELLOWS DISEASE OF CELERY, LETTUCE, AND OTHER PLANTS, TRANSMITTED BY CICADULA SEXNOTATA (FALL.). Hilgardia 3: [543]–582, illus.
(209) ———
1930. CARROT AND PARSLEY YELLOWS TRANSMITTED BY THE SIX-SPOTTED LEAF-HOPPER, CICADULA SEXNOTATA (FALL.). (Phytopath. note.) Phytopathology 20: 920–921.
(210) ———
1932. TRANSMISSION OF CARROT, PARSLEY, AND PARSNIP YELLOWS BY CICADULA DIVISA. Hilgardia 7: [163]–179, illus.
(211) ——— and FRIETAG, JULIUS H.
1938. WESTERN CELERY MOSAIC. Hilgardia 11: [493]–558, illus.
(212) SMITH, ERWIN F.
1920. AN INTRODUCTION TO BACTERIAL DISEASES OF PLANTS. 688 pp., illus. Philadelphia and London.
(213) THOMPSON, R. W.
1934. A PRELIMINARY REPORT ON THE CONTROL OF TARNISHED PLANT BUG, LYGUS PRATENSIS L. IN CELERY. Ent. Soc. Ontario Rpt.. (1933) 64: 43–47, illus.
(214) UNITED STATES DEPARTMENT OF AGRICULTURE.
1933. BLACK-HEART OF CELERY CAN BE CONTROLLED. U. S. Dept. Agr. Yearbook 1933: 339–340.
(215) WEBER, GEORGE F.
1931. BLIGHT OF CARROTS CAUSED BY SCLEROTIUM ROLFSII, WITH GEOGRAPHIC DISTRIBUTION AND HOST RANGE OF THE FUNGUS. Phytopathology 21: 1129–1140, illus.
(216) WELLMAN, F. L.
1934. IDENTIFICATION OF CELERY VIRUS 1, THE CAUSE OF SOUTHERN CELERY MOSAIC. Phytopathology 24: 695–725, illus.
(217) ———
1934. WEATHER CONDITIONS ASSOCIATED WITH SEASONS OF SEVERE AND SLIGHT CELERY EARLY-BLIGHT EPIDEMICS IN FLORIDA. (Phytopath. note.) Phytopathology 24: 948–950.
(218) ———
1935. DISSEMINATION OF SOUTHERN CELERY-MOSAIC VIRUS ON VEGETABLE CROPS IN FLORIDA. Phytopathology 25: 289–308, illus.
(219) ———
1935. THE HOST RANGE OF THE SOUTHERN CELERY-MOSAIC VIRUS. Phytopathology 25: 377–404.
(220) ———
1937. CONTROL OF SOUTHERN CELERY MOSAIC IN FLORIDA BY REMOVING WEEDS THAT SERVE AS SOURCES OF MOSAIC INFECTION. U. S. Dept. Agr. Tech. Bul. 548, 16 pp., illus.
(221) WHITE, R. P.
1926. RHIZOCTONIA CROWN ROT OF CARROTS. Phytopathology 16: 367–368, illus.
(222) WHITE-STEVENS, R. H.
1937. SOME CELLULAR CHANGES IN CELERY DURING FREEZING AND FROST HARDENING. Amer. Soc. Hort. Sci. Proc. (1936) 34: 570–576, illus.
(223) WILSON, J. D., and NEWHALL, A. G.
1930. THE CONTROL OF CELERY BLIGHTS. Ohio Agr. Expt. Sta. Bul. 461, 30 pp., illus.
(224) WINTERS, R. Y.
1909. REPORT OF ASSISTANT IN BOTANY. Fla. Agr. Expt. Sta. Ann. Rpt. 1938: XCVI–CIV.
(225) WORMALD, H.
1914. A BACTERIAL ROT OF CELERY. Jour. Agr. Sci. [England] 6: [203]–219, illus.
(226) ———
1917. THE CELERY-ROT BACILLUS. Jour. Agr. Sci. [England] 8: [216]–245, illus.

U. S. GOVERNMENT PRINTING OFFICE: 1941